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*SECTION OF PHYSICS AND CHEMISTRY**

THE STRUCTURE AND CONSTITUTION OF MUCUS**

By Harold Kwart and Victor E. Shashoua†

Department of Chemistry, University of Delaware, Newark, Del.

Mucous substances comprise one of the fundamental classes of polymers constituting the animal body. They are distributed[1] in such varied materials as gastric mucus, salivary mucus, bovine cornea, blood group substances, bone, and connective tissue. Previous workers have concentrated their investigations on the hexosamine nature of a mucopolysaccharide fragment of mucous materials. In our present work a study of the mucus from the large marine snail, *Busycon canaliculatum* Linnaeus‡, was initiated in an effort to investigate the existence of a mucoprotein-mucopolysaccharide complex, and to elucidate some details of its structure and constitution. When the term mucus is employed in this report, no implication of an identity with mucous materials from other sources is intended.

One of the outstanding features of any mucous material is its high hydration property. In *Busycon* mucus, a solution which contains only 0.3 per cent organic solids exhibits a viscosity index in the range of 60 to 100 centipoises. The binding of such immense proportions of salts and water into the composition of mucus is indeed a remarkable property, and the manner of binding bears some explanation. Moreover, it is known that mucus consists of a mucoprotein bound to a mucopolysaccharide in a physically separable manner. What is the mode of this linkage? Is this a general way in which carbohydrates are bound to proteins? These are some of the questions relating to the structure and constitution of mucus that will be considered in this paper.

Preparation and Properties of Mucus

The mucus was obtained from the hypobranchial gland according to the method of Ronkin.[2] The hypobranchial glands are excised from the snail and allowed to stand in 0.5 M NaCl. The mucus is secreted as a viscous mass that is rolled off on an applicator stick and deposited in an ice-cooled beaker. By this means it is possible to extract 100 ml. of mucus from 50 snails. The fresh mucus is then purified by centrifugation to re-

*The Section of Biology held a meeting on April 8, 1957, at which a paper entitled "The Cellular Slime Molds: A Tool for the Study of Differentiation" was presented by John Tyler Bonner. No copy of this paper has been received.

**This paper, illustrated with lantern slides, was presented at a meeting of the Section on April 2, 1957

† Present address: E. I. du Pont de Nemours & Co., Inc., Wilmington, Del.

‡ This animal is available at Wood's Hole, Mass., and in Delaware Bay during most of this year.

move cellular debris and the resultant viscous, blue, opalescent solution is used for study.

The freshly extracted mucus exhibits the two distinct properties of "elastic recoil" and "stress fibrillation."[3] The first effect is lost by homogenization under shear, by standing for 5 to 6 hr. at room temperature, and by storage in the refrigerator for a period of more than one week. This property can be characterized as the unwinding tendency of mucus after it is stirred in a given direction with a glass rod. The stress fibrillation property of mucus is characterized by the ability to form fibers under stress orientation; thus, when an applicator stick is immersed into the freshly extracted mucus solution and withdrawn, the polymer solution of about 3.5 per cent solids (3.3 per cent salt and 0.2 per cent polymer) forms a filament by extruding the solvent into small spherical beads along the fiber length. Fibers as long as 10 in. could be formed by this method with a diameter of 1 to 2 μ.

A Study of the Extraction of Mucus

In its natural state mucus is secreted into sea water and behaves as a lubricant in a medium of high ionic strength. This brings up the question as to whether salts are necessary for the evolution of mucus and whether there is an optimum *p*H value at which the secretion is evolved. TABLE 1 summarizes the results of the extraction with different buffered solutions. It is seen that no mucus is evolved in the acidic *p*H region where the gland becomes stiff and coiled, but that there is copious evolution at around *p*H 7.2 to 8, which is close to the isoelectric point at *p*H 7.7.

TABLE 1

THE EFFECT OF *p*H ON THE EVOLUTION OF MUCUS

*p*H	*Condition of gland*	*Evolution of mucus*
3.0	stiff	0
4.0	stiff	0
6.0	stiff	1
6.6	soft	2
7.2	soft	4
8.0	soft	3

Note: The highest number is proportional to the greatest yield of mucus.

TABLE 2 shows the results of extraction of the hypobranchial glands of *Busycon* with different salt solutions. A glance at the table shows that aqueous solutions of salts of monovalent cations give rise to the copious evolution of mucus, but that all divalent and polyvalent cationic salts give no mucus evolution. Furthermore, all anions, with the excep-

tion of oxalate, appear to have no influence on the mucus secretion. In addition, all polyvalent cations cause the gland to become stiff and coiled with the exception of barium and calcium ions. Although the salts of the latter ions do prevent the evolution of mucus, they nevertheless allow the gland to remain soft, and cause the mucus-bearing layer of gland to become detached from the rest of the tissue. The significance of this result may become apparent in a later section of this report.

TABLE 2

EXTRACTION OF MUCUS WITH DIFFERENT AQUEOUS SALT SOLUTIONS

Solvent	*Condition of gland*	*Evolution of mucus*
Distilled H_2O	soft	0
0.5 M NaCl	soft	4
0.5 M LiCl	soft	3
0.5 M KCl	soft	3
0.5 M NH_4Cl	soft	4
0.5 M $(NH_4)_2SO_4$	soft	4
0.5 M NH_4Ac	soft	4
0.5 M NaAc	soft	4
0.5 M KCNS	soft	3
0.5 M BaCl	soft	0
0.5 M $CaCl_2$	soft	0
Saturated $(NH_4)_2(COO)_2$	soft	0
Saturated $Na_2(COO)_2$	soft	0
0.5 M $CuCl_2$	stiff and coiled	0
0.5 M $FeCl_3$	stiff and coiled	0
0.5 M $Al_2(SO_4)_3$	stiff and coiled	0

The highest evolution of mucus corresponds to the highest number.

The Polyelectrolyte Nature of Mucus

A polyelectrolyte has the properties of both polymers and electrolytes. Electrochemically, the polymers comprise three classes: polyacids, polybases, and polyampholytes. The polyampholytes are characterized by the fact that they contain both acidic and basic ionizable sites in heterogeneous fashion along the polymeric chains. Fuoss[4] and Katchalsky[5] have described the many features of polyelectrolytes and have pointed out their importance in biologically active substances such as chondroitin sulfuric acid and hyaluronic acid. The simplest method for demonstrating that a given substance is a polyelectrolyte is through a study of its viscosity behavior. Here a plot involving the reduced specific viscosity according to the Huggins equation (1) does not show the expected linear relation

$$\frac{\eta sp}{C} = [\eta] + K[\eta]^2 C \qquad (1)$$

where ηsp is specific viscosity, $[\eta]$ is the intrinsic viscosity, C is the concentration, and K is a constant.[6] FIGURE 1 shows the application of the Huggins equation to a freshly extracted mucus sample that had been dialyzed at 0° C. to remove all its salts. Here it is seen that the nonlinear plots characteristic of polyelectrolytes are obtained. It has been suggested[4] that this behavior is a consequence of the effect of dilution producing a change in the extent of counter-ion binding by the polyelectrolyte spine. The addition of a simple electrolyte, such as sodium chloride, to polyelectrolytes has been described as preventing this abnormal dilution effect, for under such circumstances good adherence to the Huggins equation has been observed. Therefore, the viscosities of fresh mucus in 0.5 N NaCl were studied. FIGURE 2 shows the plot of the Huggins equation for such a system. It is seen again, however, that nonlinear relations are obtained (FIGURE 2). This type of behavior has been shown by Wall[7] to be due to a polyampholyte character where the nature and extent of the counter-ion binding is not kept constant on dilution. FIGURE 3 shows a plot of the Huggins equation for mucus extracted with 0.5 M $CaCl_2$. Here it is seen that a good straight-line relationship is obtained, indicating polyampholyte character, with Ca^{++} forming an integral part of the structure of mucus. It will be shown that this observation correlates very well with the presence of calcium ions in the structure of mucus as determined by ash analysis.

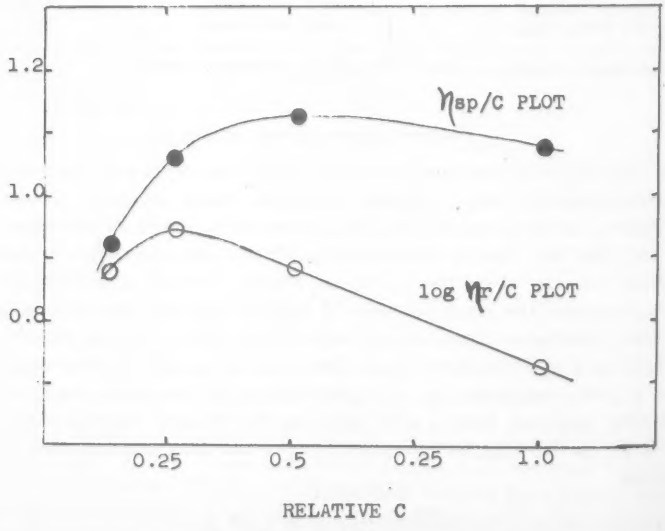

FIGURE 1. Viscosity of fresh mucus in H_2O.

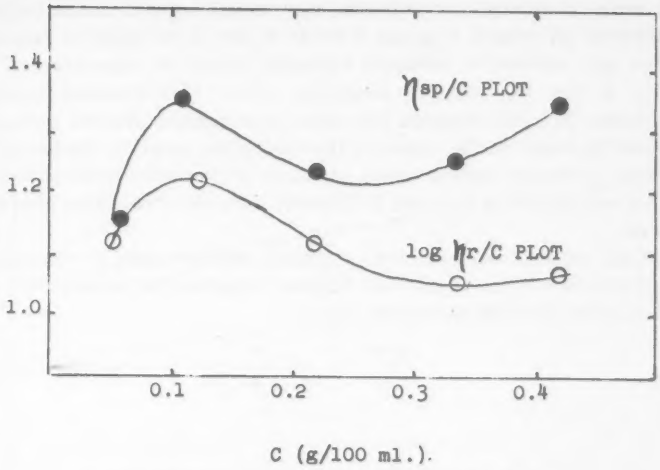

FIGURE 2. Viscosity of fresh mucus in 0.5 N NaCl.

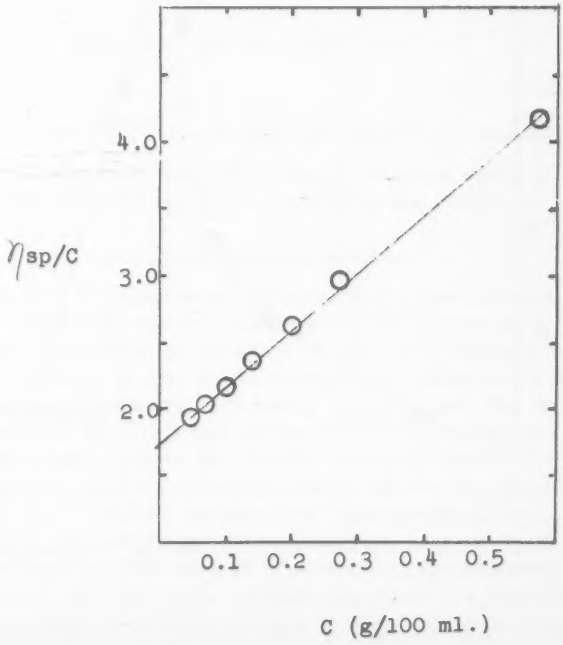

FIGURE 3. $[\eta]$ of mucus in 0.5 M $CaCl_2$.

A series of viscosity experiments was carried out with mucus buffered to different *p*H values. FIGURE 4 shows a plot of the specific viscosity versus *p*H, wherein a maximum viscosity occurs at approximately *p*H 7.2 to 8; that is, near the isoelectric point. This evidence tends to corroborate an ionic character for mucus and suggests that the molecules are most hydrated in the region of the isoelectric point. A number of the intrinsic viscosity measurements of mucus in various citrate-phosphate buffers are plotted in FIGURE 5. Clearly, intrinsic viscosities also vary with *p*H.

A final piece of evidence from viscosity measurements is obtained by the application of the Fuoss and Strauss[8] equation for polyelectrolytes. This requires for polyelectrolytes that a

$$\frac{\eta sp}{C} = \frac{A}{1 + B\sqrt{C}} \qquad (2)$$

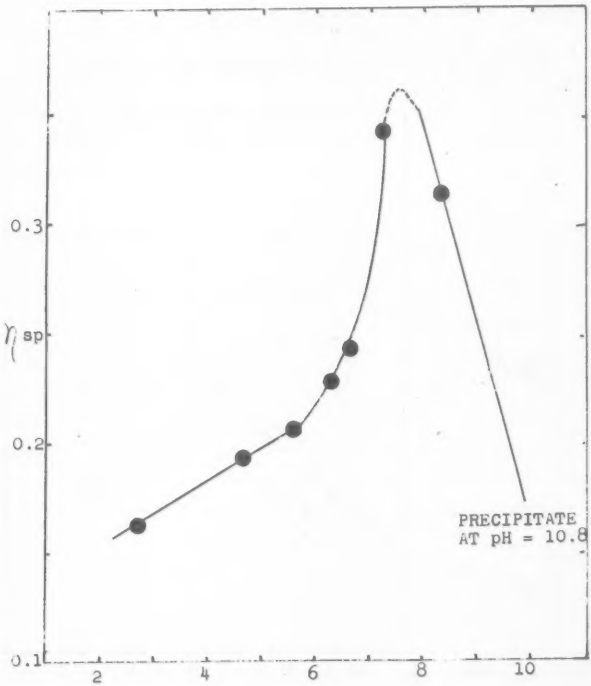

FIGURE 4. The effect of $[H^+]$ on the viscosity of mucus.

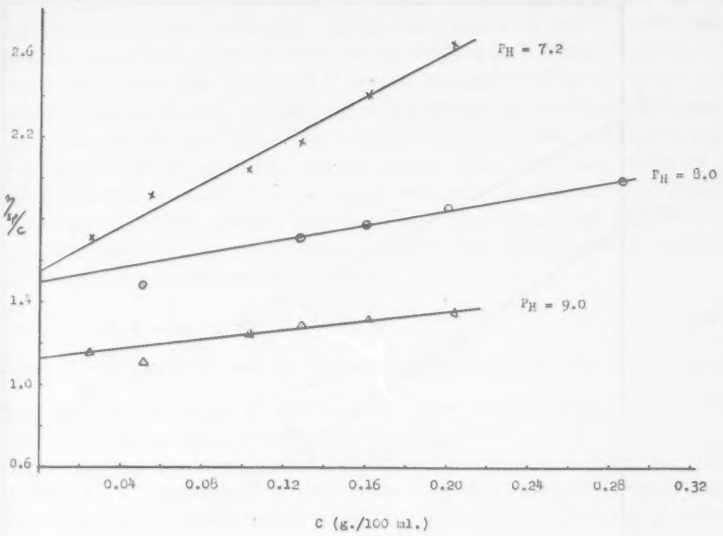

FIGURE 5. The effect of $[H^+]$ on $[\eta]$.

plot of $\frac{C}{\eta sp}$ versus $\sqrt{C}$ gives a straight line. FIGURE 6 presents a plot of the relevant data on the viscosity of mucus solutions, wherein, in the isoelectric range, it is seen that EQUATION 2 applies fairly well.

Light-Scattering Studies on Mucus

In the work with *Busycon* mucus, light-scattering measurements served, essentially, a threefold purpose: (1) the angular dissymmetry method was used in order to determine the particle size; (2) the 90° turbidity method was applied to obtain an estimate of the molecular weight of the mucus (that is, mucopolysaccharide-mucoprotein complex) and the isolated mucopolysaccharide fractions; and (3) the 90° turbidity method was used to obtain additional evidence for the polyelectrolyte nature of mucus.

Debye[9] has described a very suitable method for the determination of particle size in biological systems from light-scattering data. This method requires only knowledge of the refractive index of the solvent and the wave length of light used. In its application, the scattered light intensities at 45° and 135° to the incident light beam (λ = 5460 Å.) are measured. From these intensities the value Q is calculated according to EQUATION 3.

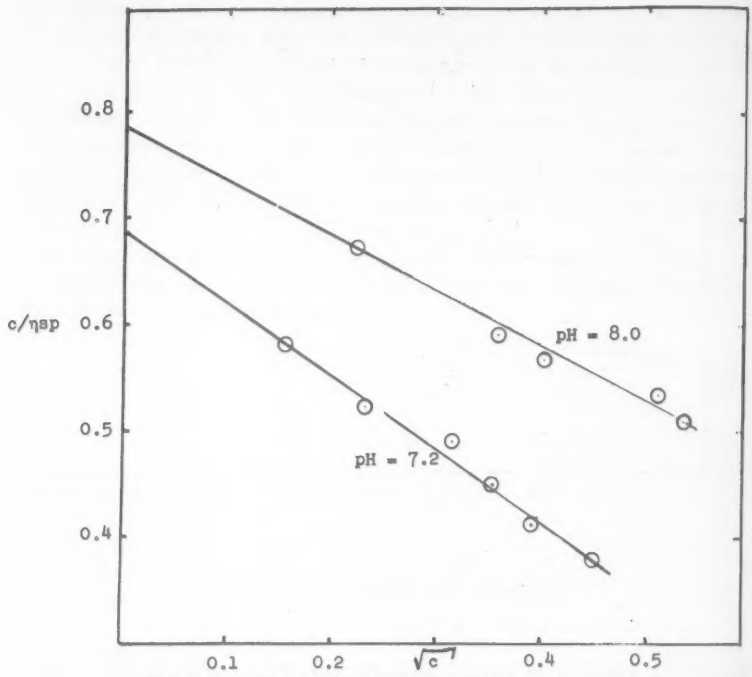

FIGURE 6. Fuoss equation applied to mucus.

$$Q = \frac{\left(\frac{R_{45^\circ}}{R_o}\right)_M - \left(\frac{R_{45^\circ}}{R_o}\right)_S}{\left(\frac{R_{135^\circ}}{R_o}\right)_M - \left(\frac{R_{135^\circ}}{R_o}\right)_S} \qquad (3)$$

where R denotes the reading at the specified angle, and the suffixes M and S represent the values for mucus and pure solvent, respectively. A plot of Q versus the relative concentration of the mucus is then constructed. Extrapolation of the linear plot to infinite dilution gives Q_o, which is used to estimate the particle size. This computation involves a knowledge of the particle shape. It is necessary to know whether the mucus molecules are spherical, random coil, or rodlike in character. The rodlike possibility was eliminated from the study of electron micrographs of homogenized samples of *Busycon* mucus. Therein, it was found that the primary mucus particle was approximately 200 Å. in diameter, and that there were occasional aggregates approaching 1000 Å. particles. Of the two remaining possible shapes the random coil configuration was eliminated by the following considerations.

Katchalsky[5] has discussed the now well-established idea that random coil polyelectrolyte molecules change their shape to rodlike molecules in dilute solution, when the counter-ion binding is not kept constant. In accord with this idea, a plot of Q versus concentration for a random coil polyelectrolyte, studied in distilled water, should be anything but linear. Since the mucus data do afford a linear plot, we have concluded that random coil-shaped particles do not occur here. The only model of a polyelectrolyte particle that is consistent with maintenance of shape with dilution is the sphere. Based on this conclusion, the diameter of spherical particles of mucus was computed from the Q_o value employing nomographs based on EQUATION 4

$$I_\theta = [3/X^3 (\sin X - X \cos X)]^2 \tag{4}$$

where $X = \frac{2\pi d}{\lambda} \sin \theta/2$ and d = diameter of the sphere, I_θ is the scattered light intensity at angle θ, and λ is the wave length of light used. The details of this method are discussed by Debye.[10 a,b]

The average swollen particle diameter of mucus was found to be 1800 to 2000 Å. A comparison of this value with the electronmicrograph result (200 Å.) for dried mucus shows an increase in diameter of nine- to tenfold, corresponding to about 800- to 1000-fold swelling in volume upon hydration. The enormous capacity of mucus for water, therefore, correlates with the unusual spherical shape of the polyampholytic structure.

The molecular weight of mucus was determined by the 90° turbidity method. Here Debye's[11] method was applied to calculate the molecular weight according to EQUATIONS 5 and 6

$$H \frac{C}{\tau} = \frac{1}{\bar{M}_w} + 2BC \tag{5}$$

where C = concentration, τ = turbidity, $\bar{M}_w$ = average molecular weight, B = a constant, N_o = Avogadro's number, η_o = refractive index of solvent, λ = wave length of light used in measurement, and $\frac{d\eta}{d_c}$ = the refractive index gradient of the solution.

$$H = \frac{32\pi^3}{3N_o} \frac{\eta_o}{\lambda 4} \left(\frac{d\eta}{d_c}\right)^2 \tag{6}$$

A plot according to EQUATION 5 exhibits a linear relation between c/τ and c with intercept $1/\bar{M}_w$ (FIGURE 7).

It is sufficient to say here that the turbidity measurements were corrected for destructive interference caused by the greater forward than backward scattering of light according to the method of Debye and his colleagues.[11]

The molecular weights were found to be extremely high. Values of 2.7

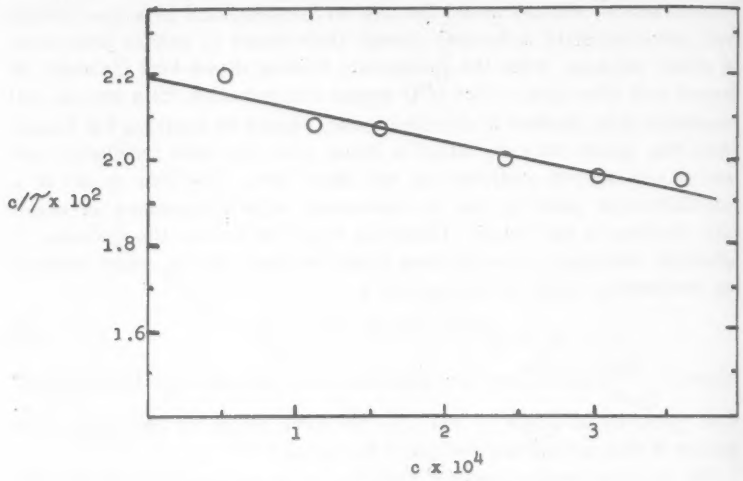

FIGURE 7. Molecular weight determination.

to 3.5×10^7 were obtained for the mucopolysaccharide-mucoprotein complex of mucus, while the isolated mucopolysaccharide fraction gave a molecular weight of 2.5×10^6.

An additional piece of evidence for the polyelectrolyte nature of mucus was obtained from a consideration of the light-scattering data. In FIGURE 7, the plot of c/τ versus c gives straight lines with negative slopes. This is contrary to experience with all standard polymers that give positive slopes in such plots, and is characteristic of polyelectrolytes as described by Doty and Edsall.[12]

The molecular weight results can be used to calculate a particle size for the dry mucus molecules in the dry state, using EQUATION 7

$$\bar{M}_w = 4/3\ \pi\ r^3\ pN_o \qquad (7)$$

where r = the radius of the spherical particle, p = the density and N_o = Avogadro's number. Using a density value of 1.2 for dry mucus, a diameter of 208 Å. is calculated. This is quite similar to the electron micrograph results, and provides additional evidence for the immense swelling that occurs with hydration of the mucus particles as demonstrated by the angular dissymmetry measurements.

The Chemical Composition of Mucus

An examination of the general composition of mucus is shown in TABLE 3. It is seen that various samples of mucus showed different ash content analysis. In sample 2 it is seen that extraction with 0.5 N NaCl

TABLE 3

ASH ANALYSIS OF MUCUS

Expt.	Extracting solvent	Mucus solids content	Ash content	Composition	
				Organic	Inorganic
1	0.5 N NaCl	0.25 per cent			
2	0.5 N NaCl	0.40 per cent	11.9 per cent	88 per cent	12 per cent
3	0.5 M $(NH_4)_2$ $\bar{A}c$ (dialyzed)	0.69 per cent	4.85 per cent	95 per cent	5 per cent
4	0.5 M $(NH_4)_2$ $\bar{A}c$ (dialyzed)	1.7 per cent	4.6 per cent	95 per cent	5 per cent

before dialysis gives approximately 12 per cent ash content as opposed to 5 per cent ash content for a dialyzed sample extracted with 0.5 M ammonium acetate. This would indicate the presence of a substantial inorganic component in mucus, a part of which is very firmly bound in the organic structure and a part of which is readily removed by dialysis.

An examination of this ash by means of emission spectroscopy showed the presence of the two elements, sodium and calcium, in major amounts in addition to a large number of trace elements that would be accounted for by the normal distribution of elements in sea water. TABLE 4 shows the results of flame photometric studies[13] of the ash of various samples of dialyzed mucus. The data make it very evident that sodium is present in samples extracted with calcium acetate and that calcium is present in samples extracted with sodium chloride. In sample 4, mucus extracted with 0.5 M ammonium acetate was subjected to a series of dialysis experiments. The results for the undialyzed sample show that the mol ratio of sodium to calcium was 14 but, as dialysis progressed, this ratio went up to 19 at a point where nearly half the inorganic content has been dialyzed off. The completely dialyzed sample had a sodium-calcium mol ratio of 1 with about a tenth of the total initial ash. This is taken to indicate that both sodium and calcium ion are gradually lost by dialyzing mucus solution, possibly through a hydrolytic process. The fact that a maximum mol ratio of sodium to calcium was reached somewhere during the dialysis process suggests that calcium ion is lost much more readily under these conditions. This can be accounted for by the structural hypothesis for mucus (see below).

The Organic Composition of Mucus

Karl Meyer[1] has pointed out the generality that mucuslike substances are composed of mucopolysaccharides complexed with mucoproteins. *Busycon* mucus is apparently no exception to this general rule. Bacila and Ronkin[14] have noted that *Busycon* mucus can be fractionated into

TABLE 4

ANALYSIS FOR NA AND CA

Sample No.	Extracting solvent	Amount of mucus*	Treatment	Analysis		Amount of mucus ash	Mol ratio
				Ca^{++}	Na^{+}		Na^{+}/Ca^{++}
1	0.5 N NaCl	0.0537 gm.	not dialyzed	550×10^{-6} gm.		6.4×10^{-3}	
2	0.5 M $Ca\bar{A}c_2$	----	not dialyzed		140×10^{-6} gm.		
3	0.5 N $NH_4\bar{A}c$	39 ml.	dialyzed	174×10^{-6} gm.	99×10^{-6} gm.	1.7×10^{-3} gm.	1
4	0.5 N $NH_4\bar{A}c$	50 ml.	not dialyzed	466×10^{-6} gm.	3726×10^{-6} gm.	21×10^{-3} gm.	14
4A	0.5 N $NH_4\bar{A}c$	50 ml.	dialyzed	157×10^{-6} gm.	1710×10^{-6} gm.	13.4×10^{-3} gm.	19

*The weight of mucus was determined by a difference in the content of the sample and the solvent solids.

two components: a protein that gives a positive biuret test, and a carbohydrate that gives a positive Molisch test. These results have been confirmed and amplified as shown in the following discussion.

The fractionation procedure consists of stirring a mucus solution with an equal volume of chloroform for 16 hr. in the presence of an electrolyte, that is, 0.5 NaCl. During this period the protein precipitates as a denatured material at the interface of the two liquids. It is separated by centrifugation and purified by dialysis. Through such repeated chloroform treatments it was found that the mucoprotein could be completely removed from the mucopolysaccharide fragment to give an essentially protein-free mucopolysaccharide fraction. This fraction gives a positive Molisch test and a negative biuret test, while the protein fraction gives the opposite results. A study of the conditions for the mucus fractionation by the chloroform technique showed that an added electrolyte was necessary in order to cause a separation of the protein-carbohydrate complex; that is, no fractionation occurred in distilled water. Furthermore, the type of salt in the aqueous phase did not appear to be critical, since ammonium chloride, ammonium acetate, and ammonium sulfate were equally as effective as sodium chloride. Apparently, the protein is denatured by the chloroform only in the presence of an electrolyte.

Before further study each fraction was purified by dialysis and lyophilized. TABLE 5 shows an elemental analysis for carbohydrate and the protein fraction. Obviously, the two fractions are quite distinct, and the total ash content of the mucus is associated with the carbohydrate fraction rather than with the protein. The ratio of the carbohydrate to the protein fraction in mucus was found to be about 40/60. Infrared analysis confirmed the purity of the isolated fractions. The absorption bands characteristic of NH_2, SO, SO_2, and OH at 6.15 μ, 8.15 μ, 9.4 μ, and 2.9 μ, respectively, were found in the mucopolysaccharide fraction. Both the carboxyl and amide bonds were conspicuously absent in the mucopolysaccharide fragment. The infrared absorption spectrum of the protein fraction was distinctly different from the carbohydrate fraction.

TABLE 5

ELEMENTAL ANALYSIS

	Carbohydrate fraction	*Protein fraction*
C	23.4 per cent	47.9 per cent
H	4.6 per cent	6.6 per cent
N	4.9 per cent	11.3 per cent
S	7.2 per cent	--
Ash total	12.2 per cent	0 per cent

The addition of barium chloride to a solution of the polysaccharide fraction acidified with HCl gave an immediate precipitate of barium sulfate, indicating the presence of ionic sulfate groups in this polymer. This was confirmed by ashing the carbohydrate fraction and observing sulfur in the ash. The close correspondence of the direct sulfate analysis and the sulfur content of the ash provides additional support for the conclusion that all the sulfur present in the carbohydrate is combined as sulfate.

Bacila and Ronkin[14] have shown that the carbohydrate fraction is based on galactosamine and glucosamine. We have confirmed this observation both by paper and by ion-exchange chromatographic analysis to the extent of finding glucosamine.

Chromatographic Analysis of Mucoprotein Fraction

The amino acid composition of the mucoprotein fraction was determined both by paper chromatography and ion-exchange chromatography on the hydrolysates. The technique employed was essentially that described by Block, Durum, and Zweig.[15] The protein was hydrolyzed by heating a 20 to 40 mg. sample at 120° C. with 20 cc. of 6 N HCl for periods of 48 hr. The hydrolysate was then evaporated to dryness and the drying completed over KOH in a vacuum desiccator.

TABLE 6 shows a quantitative analysis of the mucoprotein fraction obtained by ion-exchange chromatography.

The method of analysis was essentially that described by Moore and Stein.[16] Among the 17 different amino acids ascertained in the composi-

TABLE 6

QUANTITATIVE ANALYSIS OF THE MUCOPROTEIN

Amino acid	*Mol*	*Weight per cent*
Cysteic acid	1.8	1.88
Aspartic acid	10.0	11.27
Threonine	5.9	11.23
Serine	5.0	9.09
Glutamic acid	12.0	15.28
Proline	2.70	2.69
Glycine	7.12	4.62
Alanine	7.23	5.57
Valine	6.00	6.08
Isoleucine	4.39	4.98
Leucine	8.13	9.22
Tyrosine	4.04	6.31
Phenylalanine	1.05	1.53
Histidine	2.28	3.41
Ornithine	0.30	0.35
Lysine	3.41	4.32
Arginine	1.42	2.14

tion of the protein fraction, the mol fraction of the acidic amino acids (glutamic and aspartic) is approximately three times that of the basic amino acids (histidine, arginine, lysine, and ornithine).

Structure and Synthetic Analogues of Mucus

It is possible to postulate a structure for mucus from the evidence presented above. Essentially, there are four structural features that must be fitted together: (1) a mucopolysaccharide fragment based on a hexosamine; (2) a sulfate ion attached to the mucopolysaccharide; (3) a protein fraction with a predominance of acidic groups; and (4) the two inorganic ions sodium and calcium. In the polysaccharide fraction, the sulfate ion has two possible points of attachment; namely, on the C_2 position as an amine bisulfate or on the C_6 position as a sulfate ester of the hexosamine. In hyaluronic acid the latter was shown by Meyer[17] to be the case. In the polysaccharide moiety of *Busycon* mucus the amine function is free of acetylation as judged by a lack of amide in the IR spectrum, and the sulfate is clearly a labile ionic linkage, in distinct contrast to that in hyaluronic acid. Both of these observations are, however, quite consistent with the C_2-amine bisulfate assignment.

We proceed from this conclusion to the assumption that the mucopolysaccaride bisulfate fraction is complexed through (divalent) calcium to the mucoprotein by an ionic link as shown in FIGURE 8. The sulfate

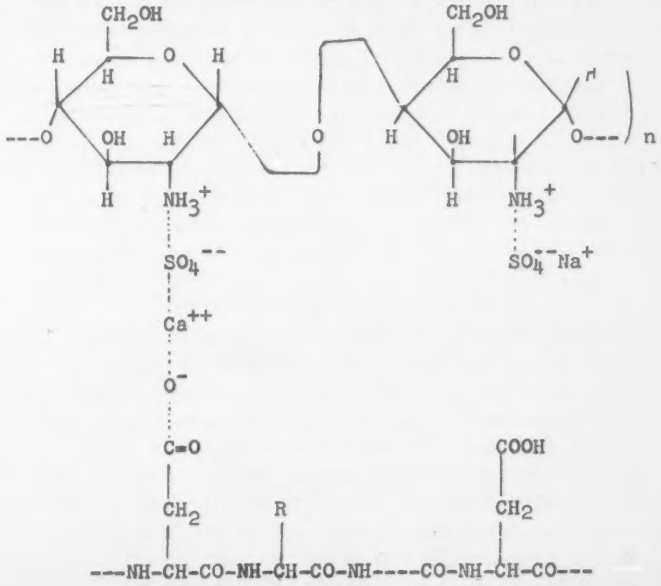

FIGURE 8.

group is probably linked through the calcium atom to a carboxyl group from either a glutamic acid or an aspartic acid residue in the protein. The polysaccharide fraction also has a number of sulfate groups with sodium as one counter-ion and the C_2-amine group (as ammonium) the other.

Evidence for the Structure Proposed

The structure in FIGURE 8 appears to agree quite well with the experimental evidence. It is of interest to list some of the observations on mucus and show how they are explained by this structure.

(1) It is found that, on fractionation, all the inorganic ions become attached to the mucopolysaccharide fraction, since the protein shows no ash. This is well explained by the fact that the bond between the sulfate and the calcium ion is less susceptible to hydrolysis than the bond between the calcium and the carboxylate ion(s).

(2) It is found that mucus can be extracted by salt solutions containing any type of monovalent cations, whereas no extraction is possible by salt solutions of divalent cations. This is consistent with the fact that monovalent cations can exchange with the calcium and hence decrease the branched or cross-linked nature of the mucus to give more solubility, whereas divalent ions can only increase the degree of cross-linking by the same mechanism to make the mucus less soluble.

(3) It is found that fractionation by the chloroform method is possible only when there is a univalent metal ion in distilled water. This is in agreement with the observation that the protein, in an aqueous solution, is well bound to the solubilizing carbohydrate fraction. In an aqueous salt solution, however, ion exchange can take place to release the protein, which then denatures on contact with chloroform.

(4) It is found that the intrinsic viscosity of mucus is a maximum in calcium chloride solution. This might well be due to the lack of any hydrolysis or ion exchange of the native calcium in the presence of excess calcium ion, so that the natural structure of mucus is preserved.

A Test of the Structure Proposed

In an attempt to test the structural hypothesis, a sample of mucus was placed in a 5 per cent versene (di-sodio-ethylene diamine tetra-acetate) solution at *p*H 4.2 for 24 hr. This was then dialyzed for 3 days against a versene solution and for an additional 3 days against distilled water. The dialysis was observed periodically throughout its course, and it was not until a substantial amount of versene had been complexed and removed by dialysis that a fibrous precipitate formed in the dialysis bag. This was centrifuged from the mixture and was found to give a positive biuret test, indicating protein character. Moreover, the mucoprotein in this instance was not denatured, since it was quite soluble in water. A check on this

observation was carried out by flame photometric analysis. The data proved that more than half of the calcium had been complexed by the versene when the protein precipitation was observed at a *p*H of 4.2.

A Comparison of Busycon Mucus to other Mucous Materials

The essential features in the structure of mucus are an acidic mucopolysaccharide fragment linked to an acidic mucoprotein through a calcium ion. One might ask how widely this concept can be applied. A search through the literature reveals that, while a considerable amount of work has been done on polysaccharide fragments of various mucous substances, comparatively little is known about the mucoprotein fraction. However, at least part of the observations we have reported here on the composition of *Busycon* mucus may have some general application. It has been noted, for instance, that in bone mucus,[18] human plasma mucus MPI,[19] and ovomucoid[20] the acidic amino acids are present in approximately twice the molar quantity of the basic residues. In other instances, Grant's[21] report that intravenous injection of calcium inhibits the secretion of gastric mucus and the viscosimetric studies of Ogston and Stanier,[22] which produced a value of ca. 10^7 for $\bar{M}_w$ of synovial fluid, resemble observations and magnitudes we have reported here for *Busycon* mucus.

Synthetic Analogues of Mucus

In the most general sense a synthetic analogue of mucus must have two polyacidic fragments linked by calcium ion, and it must be able to give the viscosity properties of mucus. It should be a polyelectrolyte and should display the two curious properties of "elastic recoil" and "stress fibrillation." In a first attempt to simulate these properties a 3 per cent solution of polyacrylic acid in water was mixed with calcium carbonate. Not only were elastic recoil and stress fibrillation behavior noted for this solution, but, in fact, an addition of 0.5 N sodium chloride caused a decrease in these properties in a fashion analogous to mucus solutions.

Another series of experiments were carried out with a view to obtaining a natural analogue of mucus. Thus, chitosan (poly-2-aminoglucose) was degraded with acid to a water-soluble fraction. The addition of sodium bisulfate and admixture with a solution of polyacrylic acid that had been neutralized with soda lime or its equivalent produced a solution that exhibited many of the overt physical characteristics of mucus as noted above.

Acknowledgment

We thank R. Ronkin of the Biology Department, University of Delaware, Newark, Del., whose original suggestion evoked our interest in this problem and Paul Hamilton of the Alfred I. du Pont Institute of the

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SECTION OF ANTHROPOLOGY

TIV MARKETS*

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One of the most astonishing aspects of West African ethnographic literature is that it contains no adequate study of marketing in an area noted for having the most highly developed indigenous markets in the primitive world. This paper is a preliminary and partial report on a study now being completed that will begin to mend this situation and that deals with the social organization and the economic functions of markets among the Tiv of central Nigeria**. The Tiv are an agricultural people numbering about 800,000. They speak what is commonly called a semi-Bantu language. Anthropologically, the most interesting aspects of their culture are their jural system, their complex system of exchange marriage, and their system of witchcraft beliefs. Economically, their situation is simple: they live by subsistence farming, and they exchange their produce either for other goods, or for money with which they may pay tax and bridewealth or buy imported goods.

Tivland is covered by a network of markets. In fact, it might be more accurate to say that it is covered by two such networks. One is political in nature: markets provide a channel for political activity. The other is economic in nature and concerns the flow of goods into and out of the country. The two networks do not coincide.

These two networks are consequent to the complex nature of Tiv markets:

(1) Markets have what might be called an aspect of sociability. People go to markets to meet one another, to drink, to keep appointments, or just to have fun. Which markets the people of any given neighborhood attend for such purposes depends upon the size of the market, lineage position, and distance.

(2) Markets have what might be called an aspect of sacredness. The days of the five-day "week" take their names from markets, and hence markets have a cosmographic aspect. Some markets are consecrated, and hence have a specifically ritual aspect. This aspect overlaps with yet another: the jural. Those different lineages that were concerned in the consecration of a market are thereby bound to settle their differences peaceably and by arbitration instead of by fighting. Obviously, then, the political aspect of markets arises from the sociable, sacred, and jural aspects of markets.

*This paper was presented at a meeting of the Section on April 22, 1957.

**Our twenty-six months' field research among the Tiv, between 1949 and 1953, was financed by The Social Science Research Council, Inc., and The Wenner-Gren Foundation for Anthropological Research, Inc., both in New York, N. Y.

The second network, the economic, is much simpler. After all, markets are places of buying and selling. However, the markets to which a person goes specifically to trade are often not the same as those which he attends for other purposes. The Tiv know and can give the investigator a general picture of the flow of goods through the markets of their country. Tiv traders can give such information in detail. The Tiv also know and willingly explain that markets are important social and political institutions, and they readily comment that a market "good" for buying and selling need not be "good" for settling disputes.

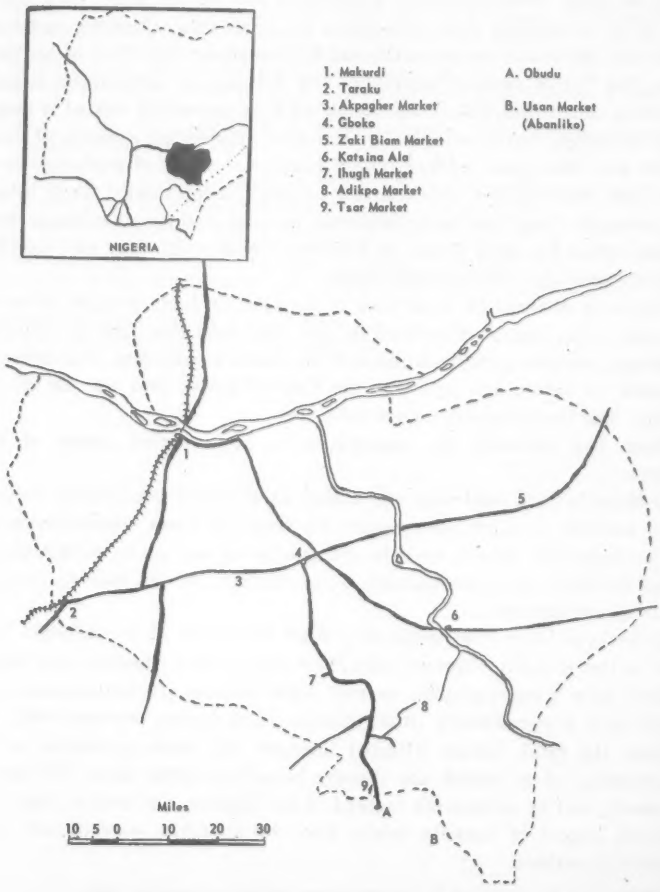

FIGURE 1. Road network and large markets in Tivland.

A description of either network obviously must start with the geographical location of market places, and it will be necessary to make constant reference to the maps. FIGURE 1 shows the road network and major markets of Tivland; FIGURE 2 illustrates a portion of southern Tivland and adjoining parts of the eastern region of Nigeria peopled by the Udam and the Uge.

The first network is composed of the markets that are attended by Tiv of a given neighborhood. It can be discovered by the unqualified question "What are the markets hereabouts?" The answer is almost always, "Today is..." and so on through a list of those markets that locally give their names to the five days of the market week.[1,2] Alternatives are seldom volunteered, and their mention – generally by a woman or a youth – is often reproved: "They are asking about markets, not about beer drinks," or, "That is not our market."

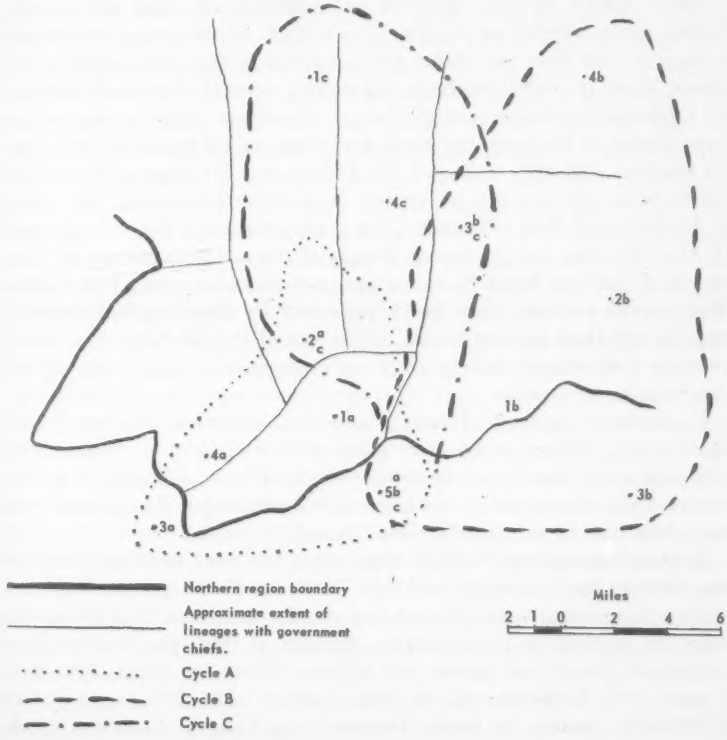

FIGURE 2. Three market cycles in southern Tivland.

In such a context, "our market" refers to the fact that each neighborhood sees itself as the center of a five-day cycle of markets that are, in fact, those most frequently attended by the people of the lineage of that area, for Tiv prefer to attend the market that is socially and spatially the closest. If the two conflict, social distance outweighs spatial distance. Thus, while both Ticha market (1a, FIGURE 2) in MbaDuku lineage area and Iyon market (1b, FIGURE 2) in Shangev lineage area were held on the same day, few MbaDuku people went to Iyon and few Shangev people went to Ticha. Each referred to the other market as "far" (*cha*). We have heard MbaDuku people who lived a good bit farther from Ticha market than from Iyon insist that Ticha was the closer. This was true in social distance. The few exceptions are also exceptions in social distance: Shangev women married into MbaDuku found it convenient to claim that Iyon market was closer; people with close kinship ties into the other lineage also found it closer or just as close.

These market cycles, centered on neighborhoods that are usually lineage areas, overlap as FIGURE 2 indicates. Almost every market has a place in more than one market cycle, central in some and peripheral in others. Thus, in central MbaDuku, the cycle a went: (1) Ticha, (2) Aichwa, (3) Chukwan, (4) Angur, and (5) Asawa. About six miles to the east in Iyon lineage of Shangev, the same days were called (cycle b): (1) Iyon, (2) MbaHura, (3) Ajio or Usan*, (4) Adikpo, and (5) Asawa. These two cycles have only one day in common. In a cycle from central Ute, north of MbaDuku and West of Shangev, the cycle c runs: (1) Pev, (2) Aichwa, (3) Ajio, (4) Ukoo, and (5) Asawa. People of Ute and Shangev met at Ajio, people of Ute and MbaDuku at Aichwa, and all three mingled at Asawa. Thus one can see how, given the Tiv preference for attending the "closest" markets and their varying social definitions of "close," the day names compose a shorthand description of the channeling of social communication from any one center.

If, however, instead of asking a general question, one asks more specifically, "Where is the best place to sell cloth?" or "Where does camwood come from?" one is given quite a different grouping of market names. This second series identifies that market network concerned with the actual flow of goods and produce through Tivland.

Scattered throughout Tivland there are a few very large markets that are hubs in the system of exchange (FIGURE 1). They both drain and supply surrounding areas through the smaller and, from this viewpoint, satellite markets in their vicinity. Markets of this type that we have mentioned already are Asawa and Adikpo. Others are Ihugh, Akpagher, Taraku, and ZakiBiam. All of these markets also serve as important distribution centers for goods imported into Tivland. They are conse-

*Usan, an Uge market, was held every tenth day and occurred in the Iyon cycle on alternate market weeks.

quently located on a motor road accessible to Ibo and Hausa traders, or on an important cross-country trade route, or at an intersection of the two. Each such market is widely known to the Tiv as a "good market" for the buying and selling of particular commodities.

Our best information on a large market of this type comes from Tsar (No. 9, FIGURE 1), a market held in MbaDuku since the spring of 1950. At that time, with the connivance of part of the Hausa colony of Obudu (in the Eastern Region), the Tiv boycotted Asawa market (A in FIGURE 1; 5abc in FIGURE 2), which they claimed had been "stolen" from them about 1915, an event that had forced the major part of the trade farther north to their own market*.

Tsar market is in MbaDuku where the motor road intersects the path taken by Bamenda and other Cameroons traders on their way to Enugu, Onitsha, and other markets in the Eastern Provinces. These traders bring cattle or hides; they return, on the same path, with imported cloth and other trade goods. Tsar is therefore a good place for the Tiv to sell produce (primarily rice, yams, cassava, and goats) and the cloth they weave themselves (preferred by the Abakaliki Ibo, who do not weave, to European cloth for its durability). These products are bought by Bamenda, by Hausa, by Udam from Obudu, and by Ibo, who come in lorries from Abakaliki, Makurdi or beyond. Tsar is also a good place for the Tiv to buy raffia and palm oil brought in from Udam and Uge country to the south, either by Tiv traders or by the Udam and Uge themselves. Tsar was a large market. To the Tiv proof of this was that as many as seven head of cattle were butchered there in one day for sale to consumers and to traders who resold the meat at smaller markets the next day. Indeed, attendance at Tsar market ran from three to five thousand people every five days on a market place about the size of an American city block.

Surrounding such major markets, or hubs, there are many medium sized markets that might be called bulking stations. They may specialize in certain kinds of produce. For example, before its discontinuance on the opening of Tsar, Ticha in MbaDuku was a market attended by five or six hundred people during its busiest season. It served as a bulking station for rice. Both men and women bought small lots of rice by the calabash-full in the smaller outlying markets. At Ticha they sold this rice, accumulated into lots of 30 or 40 lb., to Tiv middlemen. After their wives had husked it, the men then resold the hulled rice several weeks or months later to Ibo lorries at the Asawa and later at Tsar markets. The scale of these operations may be judged by the fact that one set of three full brothers in MbaDuku, whom we knew well, handled in this way several tons of rice every year.

*The cycles shown in FIGURE 2 are those prior to these events. The major markets in FIGURE 1 include Tsar. This incident will be further described in our forthcoming monograph on Tiv economics and warfare.

Not all of these specialized markets or "bulking stations" are as large as Ticha. MbaHura market, which is one of those included in cycle b centering on Iyon, was seldom attended by more than three hundred people, yet it was a place known for the purchase of guinea corn, mats, and baskets. Nor are all markets the size of Ticha in any way specialized. Nevertheless, each market cycle tends to have at least one of these medial, bulking markets. Not all cycles, however, have a large market, as Tsar, Asawa, Adikpo, and other cycles have, and only those people intent on buying and selling – or visiting kinsmen – attend a market outside their cycle. There are only two self-justifiable reasons for leaving one's work to go to market: "It is our market," for the socially closest market *should* be attended, or "I am going to buy some cotton [in eastern Tivland or in that section just south of the Benue and Katsina Ala confluence] and visit my married sister," for one must give a reason for going farther afield.

There are many smaller markets in any given cycle. Aichwa and Angur in the MbaDuku cycle are examples. Such markets may be important as "feeders" (both Aichwa and Angur were considered good places to buy rice or yams for bulking at Ticha), or they may derive their importance from the fact that they are consecrated and hence of political significance.

Scattered throughout Tivland there are many more markets too small to have a place in any five-day market week; not even the people of the immediate vicinity use their names as day names. Such markets are called "bush markets" (*kasoa shin toho*), "women's markets" or "only beer drinks." Some of these markets function as primary buying points: women come with a few yams or cassava roots and sell them, generally to other women, who resell these products at the larger markets where the prices are somewhat higher. It is from such markets, which may be gatherings of no more than fifty people for no more than two or three hours, that the larger markets are ultimately supplied with produce, and it is to such markets that the European and other foreign goods bought at the large markets eventually find their way.

In terms of trade and sociability, the overlap of Tiv markets forms the basis of their political aspect. Those particular markets appearing in two or more cycles are a meeting place of potentially hostile groups. They were thus foci either for warfare or for the settlement of interlineage disputes; it was the consecration of markets that endowed them with the latter function. In most areas the advent of the British administration has rendered this aspect of markets superfluous. However, where communication within the British administration is unreliable or slow, as is the case across the boundaries of the Northern and Eastern Regions of Nigeria, this function is not superseded, and it remains an important aspect of the consecrated Tiv markets in this area.

The consecration of a market puts it on a new footing. From that moment the market and its day are "in the nature of things." The ritual – and purpose – of consecration demand the presence of outsiders, for the avowed purpose of market magic is to assure the maintenance of peace. After the consecration of a market, breaches of peace are subject not only to the courts of the market masters or of any other secular authority, but to magical retribution as well. The form of the market magic is closely related to its purpose.

A market fetish, or *akombo* as Tiv call it, consists generally of two elements: (1) *swem* or one of the other fetishes of stature associated with justice and capable of instruction, and (2) the *ishough* tree (*Erythrina senegalensis*), which is the emblem of one aspect of the important fetish *swendegh*, associated with bloodshed. *Swem* is one of the ultimate instruments of justice.[3] If it is transgressed, a man's feet and stomach swell up and he dies. *Swendegh*, when it attacks a person who has transgressed it, takes several forms; the most common is sores. These two elements are those commonly used in Jechira lineages. Elsewhere in Tivland, one or both of them may be changed. The magic of Iyon market, for example, contained *swendegh* and *ver akombo*. The emblem of the latter is the silk cotton (*vambe*) tree, and the fetish most commonly manifests itself in those deficiency diseases common among African peoples whose staple diet is vegetable.

At the time these fetishes are set up, they are "instructed." Tiv tell us that market fetishes are established and instructed in precisely the same way as was the fetish for Iyon market bridge, rebuilt in the summer of 1950. Iyon established powerful magic at both ends of the bridge and instructed these fetishes to kill (*wua*) anyone who evacuated on the bridge, who willfully damaged it, or who shed blood on the bridge. These instructions were delivered, publicly, in a high, loud voice.

As in all forms of Tiv magic, sacrifice is necessary to make the emblems potent. The sacrifice needed for market magic is believed to consist of human beings – members of the lineage that "owns" the market, who are killed "by night" (that is, mystically, by witchcraft). All consecrated Tiv markets of which we know are said to have had such sacrifices performed for them. We believe these sacrifices to have been of the same nature as the "sacrifice" for Iyon market bridge, which we witnessed. When the bridge was almost finished, a drunken youth insisted on bathing in the river, then at flood. He drowned. The entire countryside believed that he had been mystically killed by the elders to consecrate the bridge.

The ritual establishing market magic also serves to define the "owning" lineage. Elders of that lineage invite other lineages, or tribes, to participate in what may be called a market pact. It is said today that each of the participating groups must provide a slave or a dog (for the Tiv and for many Udam a dog is the ritual equivalent of a human being)

to be buried when the emblems of the market magic are planted. In 1950 it was said that the following groups each provided a slave for the original establishment of Tsar market magic: (1) the Utisha (Tiv lineages living in the southeastern part of Tivland), (2) the Uge (the tribes known as Abanliku in the Eastern Region); (3) the Obudu, (4) the Kambi, (5) the Tukwel, (6) the Damakula, (7) the Kpanku – all of which are subtribes of the congeries of people whom the Tiv call Udam; (8) the Tiv Mbashaya, that is, MbaDuku and her companion lineages of Jechira. Three other peoples refused to send slaves, but sent dogs that were declared the equivalent. These slaves are said to have been sacrificed by the founder of the market, and their blood smeared on the *ishough* tree. The Tiv describe these as "daytime" sacrifices – that is, physical killings. Their "proof" is the statement that slaves, not lineage agnates, were killed.

The market magic is visible in the fetish emblems that are planted in the market place and that can be moved, if the market place is moved, by transplanting cuttings. The market magic is also associated with the day on which the consecration was held, and every fifth day thereafter. The Tiv say of each market, "The market is the day." Only of a consecrated market can one say, as one old man spoke of Tsar, "The day of Tsar market is in the order of things (*Aondo gba yange MbaDuku*)." An unconsecrated market can be held on any day. The day of a consecrated market cannot be changed.

Market magic has certain jural implications. It binds the members of those groups which, as groups, participated in the consecration to proper behavior in that market in perpetuity. All markets have an organization to deal with brawling, thieving, and other misbehavior. At a consecrated market, however, such an action is more heavily fined if it occurs between members of participating groups than if it is performed by others or in an unconsecrated market. For example, MbaDuku, Iyon and other Shangev lineages, and the Uge all participated in the consecration of Iyon Market. The Udam did not. We have heard a recalcitrant MbaDuku thief in Iyon market confess, give his name, and pay his fine when it was pointed out to him that the woman from whom he had stolen was an Uge. His defense was that he had thought her an Udam.

Consecrated markets have a further political aspect: they provide a time and a place, magically assured against violence, for negotiations among the participating groups. Tiv say that this was an important function of all consecrated markets in the past. Today, when the maintenance of peace and the channeling of political communication lie through the British Administration, such activities are observable only in unusual situations. Tiv markets, such as Iyon, whose member groups live on either side of the boundary between the Northern and Eastern Regions, are the best example. It is the normal situation that communication across this boundary is complex and slow. Iyon market has never

lost its position as a normal mode of communication between Shangev Tiv and the Uge. Such a market may also play the role of a neutral embassy in time of war. For example, during a war in the spring of 1950 between MbaDuku, other Kunav lineages and the Udam, the only important line of communication left open was Iyon market, which MbaDuku could attend directly and at which the Uge could represent the Udam.

In this paper we have not had space to do more than mention the existence of an internal organization of Tiv markets. It should be noted, however, that there are several types of internal organization and that these markets, if they are consecrated, correspond to Sir Henry Maine's definition of a social group as a corporation.

Nevertheless, the purpose of this paper has been to show the ways in which Tiv markets fit together into networks of markets. There is one alignment of markets for purposes of transfer and trading of goods. There is another alignment for noneconomic social purposes; this alignment is based on neighborhood and lineage. The extent to which this alignment corresponded to that of consecrated markets is now difficult to determine, as the advent of the British has rendered their political role unnecessary in all but a few border situations. Their continued importance under conditions of difficult communication and uncertain peace make it seem likely that consecrated Tiv markets served the same combination of economic, social, and political purposes that made the peace of the market of such moment in the medieval world.

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DIVISION OF MYCOLOGY

TINEA CAPITIS AND AFRICAN HISTOPLASMOSIS IN THE BELGIAN CONGO*

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Tinea Capitis in the Belgian Congo

The first important paper on dermatophytes and ringworm in the Belgian Congo was published in 1950 (R. Vanbreuseghem). The results that were presented on the Congolese dermatophytes, their relative number, and their geographical distribution have not been materially altered by subsequent researches.

Dermatophytes isolated in the Belgian Congo from native cases of tinea capitis, or of cutaneous ringworm, are mostly cosmopolitan or Afro-Asiatic species. To follow the nomenclature proposed by M. Langeron, Milochevitch, and myself, these dermatophytes should be distributed in five genera; however, to comply with American usage I shall name them after Emmons: *Trichophyton (Ctenomyces) interdigitale; Trichophyton violaceum* and its white variety *T. glabrum; Trichophyton ferrugineum* and *T. ferrugineum* var. album; *Trichophyton rodhaini; Trichophyton rubrum; Trichophyton (langeronia) soudanense; Microsporum (Sabouraudites) langeroni; Microsporum (Sabouraudites) duboisii; Microsporum (Sabouraudites) gypseum; Microsporum (Sabouraudites) rivalieri;* and *Epidermophyton floccosum.*

Most of these species are accepted by competent mycologists. The others are new and require a few words of explanation. *M. langeroni* Vanbreuseghem 1950 appears to be a very important species as it occurs in 25 per cent of the cases of tinea capitis and in many cases of ringworm of the glabrous skin. The colonies on Sabouraud agar have a pink or salmon-colored surface that is less fluffy than the surface of *M. audouini* colonies. Microconidia and macroconidia, typical of the genus *Microsporum,* are very rare; but one usually finds in old cultures large subterminal chlamydospores developing abundantly. *M. duboisii* Vanbreuseghem 1949 has been isolated only once. It is very similar to *M. gypseum,* but it forms sclerotia, which is very unusual; actually it is the only species I know among dermatophytes that does so. I described *T. rodhaini* in 1949 from a unique strain, but I found this species two or three times later. The very young colonies resemble *T. violaceum,* but

*This paper, illustrated with lantern slides, was presented at a meeting of the Division of Mycology on April 26, 1957.

they become powdery. After a good number of subcultures the fungus becomes very similar to *T. rubrum*. I wonder if it would not be wiser to consider *T. rodhaini* as a variety of *T. rubrum*. M. Ota had seen the white variant of *T. ferrugineum*. From the observation of 136 strains, I concluded that four types of colonies must be considered in the classic *T. ferrugineum*, as well as in its white variant. *M. rivalieri* Vanbreuseghem 1951, which has not been included in the statistics, will not be considered here.

At the time of the first survey our first 427 strains were distributed as follows (omitting *M. duboisii*, *T. rodhaini*, *T. interdigitale*, and *M. gypseum*, all of which had been isolated from white people only):

T. ferrugineum	175	41 per cent
T. violaceum	107	25 per cent
M. langeroni	107	25 per cent
T. soudanense	24	5.5 per cent
T. rubrum	12	3 per cent
E. floccosum	2	0.5 per cent

Neglecting *E. floccosum*, which is not a hair invader, and *T. rubrum*, which rarely invades the hair, we must admit that 68 per cent of the cases are microsporia and 32 per cent trichophytia. *T. ferrugineum*, a typical *Trichophyton* in cultures, produces in the hair a microsporic lesion.

It is remarkable indeed that dermatophytes as common as *M. canis*, *T. tonsurans*, or *T. schoenleini** have not been reported thus far. It would be interesting to discuss the geographical variations of dermatophytes and to find the cause of what I call their "regional spectrum." The Congolese *Microsporums* concentrate in the low parts of the country, but the *Trichophytons* are located chiefly in the mountains. It seems, from unpublished data, that in the hilly parts of Ruanda-Urundi only *Trichophytons* are to be found.

To appreciate the percentage of natives affected by tinea capitis, I have recently inspected 15,000 Negroes (adults and children) in the Belgian Congo and in Ruanda-Urundi. As favus is unknown in these countries, it was natural to expect tinea capitis in children only. This was true except in Ruanda-Urundi, where I observed tinea capitis of a trichophytic nature in adult women. Diagnosis was made on a clinical basis, often with the aid of ultraviolet light from the Wood lamp, and it was only admitted as conclusive after confirmation by the microscope and culture. Unless otherwise indicated the following percentages refer to children only.

Among the town dwellers in Léopoldville, a town of 325,000 natives, 6.7 per cent of the children examined were found to be affected with

*The unique isolation of *T. schoenleini* by J. Rodhain (1943) has never been confirmed.

tinea capitis, which would make, by extension, 8,200 in all. Microsporic tinea was recognized in 78.4 per cent of the children and trichophytic tinea in 21.6 per cent. Five-year-old children were chiefly affected. Eighty-seven per cent of the cases were 7 years old or less.

Among the Luluabourg town dwellers, the percentage rose to 40 per cent (55 per cent microsporia and 45 per cent trichophytia). In schoolchildren 6 to 8 years old this percentage may have attained 70 per cent. A similar percentage was obtained in town dwellers at Luebo, but in Luisa (among very primitive people living in their native villages) the percentage dropped to 2.9 per cent.

In Elisabethville the observation of 700 children showed 15 per cent of tinea capitis: 58 per cent microsporia and 42 per cent trichophytia. About the same percentage, 15.7 per cent (90 per cent trichophytia) was observed in Usumbura, the most important town of the Ruanda-Urundi, and among the natives of the valley along Lake Tanganyika. In the mountains of Ruanda-Urundi, however, the percentage rises to 32.5 per cent (100 per cent trichophytia). As already pointed out, it was in this territory that I discovered tinea capitis in adult women: 1 per cent in the valley and 10.6 per cent in the mountains. Another interesting fact is that among the 200 children of the Asiatic school of Usumbura, only one was infected.

Among the natives living in Bukavu, 20 per cent of the children were affected by tinea capitis (90 per cent trichophytia). Among the natives living in the mountains south of Bukavu, 25.5 per cent were affected in Kibila and 37 per cent in Kamanyola (this last percentage refers to schoolchildren from 7 to 14 years old). It is important to note that at the military camp in Bukavu, where the native children had good food and good surroundings, only 4.5 per cent of them were afflicted; among children 3 to 8 years old at the Atheneum school in Bukavu (attended by 147 whites, 16 natives and 12 Asiatics), not one case of tinea capitis was to be found.

Among the town dwellers of Stanleyville 5.9 per cent of tinea capitis was discovered and, in Bengamisa, 6.3 per cent (76 per cent microsporia, 24 per cent trichophytia). Less than 200 miles from Stanleyville, among the very primitive natives of Opala, not even 3 per cent were diagnosed.

What is the reason for these very different percentages of infection? It is a well-known fact that in the so-called civilized countries, tinea capitis does not affect more than 5 per cent of the children (M. Sulzberger and R. Baer, 1954, 1955). This percentage is approximately what I found in Léopoldville, Stanleyville, and Bengamisa. I propose to examine the following possibilities to explain the occurrence of the largest percentages:

Could a special virulence of some dermatophytes be held accountable? Although this may be a valuable hypothesis, it does not seem that it would be very useful in this case. For example, in Bukavu, with the

same dermatophyte flora, I discovered no cases in the Atheneum school. I found 4.5 per cent among the children of the military camp and 20 per cent among the natives dwelling in the town. Similarly, in Ruanda-Urundi I found 15.7 per cent in the valley, 0.5 per cent in the Asiatic school in the same valley and 32.5 per cent in the hills, although the dermatophytes are about the same everywhere.

Is the social and economic evolution of the natives a factor? The general conditions of hygiene and housing do not seem to explain satisfactorily the difference between the high percentage in Luluabourg and the lower percentage in Léopoldville and Stanleyville. It is among the most primitive people of Luisa and Opala that we find the lowest percentages (2.9 per cent and 3 per cent, respectively) if we make an exception of the percentages found among whites and Asiatics. If there is some truth in this hypothesis, it should be said that evolution has an unfavorable effect on the dispersion of tinea capitis.

What about the population density? This may explain why natives living in their clan villages, in larger huts dispersed in the field or along the road, are less affected than the town dwellers. However, it does not explain why the native population of Elisabethville, Usumbura, Bukavu, Astrida, and Luluabourg (respectively 15 per cent, 15.7 per cent, 20 per cent, 32.5 per cent, 40 per cent) pay a larger tribute to the disease than the populations of Stanleyville (5.9 per cent) and Léopoldville (6.7 per cent).

Can the explanation lie in the qualitative or quantitative variations of the diet? If the material conditions of life are very similar in cities, there is, nevertheless, one factor that shows very large variation, and that is the diet. I was impressed by the fact that the highest incidence of tinea capitis was found among populations where animal proteins were lacking. It is actually in Kasai and in Ruanda-Urundi – I do not include the region of Kwango, which I have not visited – that one finds numerous cases of kwashiorkor (malignant malnutrition). In Luluabourg, where tinea capitis may be found in four children out of ten, I noticed that the face or ankles were often swollen, and that a typical sign of pitting edema was present.

If there is actually a relationship between tinea capitis and a lack of proteins, this fungus disease should be found in a large percentage of children affected by kwashiorkor. Such a relation has not yet been pointed out in the textbooks on kwashiorkor (see for instance G. Gillman and T. Gillman, 1951, or H. C. Trowell, J. N. P. Davis, and R. F. A. Dean, 1954). A survey of children suffering from this disease at the Lwiro dispensary (I.R.S.A.C.) and at the Bukavu Hospital has demonstrated that out of 49 cases of kwashiorkor, 45 (or 91.8 per cent) were also affected by tinea capitis (*T. violaceum, T. ferrugineum* and *M. langeroni*).

If some native populations seem to escape the danger of tinea capitis, it is not only because their native villages are less crowded, but also

because their diet is richer in animal proteins acquired by fishing, hunting, and the raising of cattle. Although cattle abound in the hilly regions of Ruanda-Urundi, the fact that natives do not for traditional reasons eat the meat might explain their 30 per cent of tinea capitis.

The very high percentage of tinea capitis among children afflicted with kwashiorkor, the high percentage in the populations where proteins are lacking, the small percentage in the towns or native villages where proteins abound, all seem to indicate that protein deficiency is in some way linked to the disease. It is very difficult to decide whether protein deficiency is the determining factor, or if it is some other deficiency linked to it as, for instance, an avitaminosis A. Another question is to decide if the shortage of some factor acts by itself, or by the alteration that it produces in the body, particularly in the skin or its appendages. The skin and the hair are very much affected in cases of kwashiorkor. I have observed many mechanical alterations in the hair of children with kwashiorkor that might favor the penetration of the dermatophytes. Similarly, an alteration in the chemical nature of keratin, or of the glandular secretions, could be a factor.

We may accordingly conclude that a dietary factor may be linked with tinea capitis that will explain why misery and tinea capitis have always been related. Further investigation into the real nature of this factor would afford the epidemiologist, as well as the scientist in the laboratory, a way to cope with tinea capitis as a public health problem and as an experimental subject also.

African Histoplasmosis and Histoplasma duboisii

In 1952 I suggested, in Langeron and Vanbreuseghem, that an African histoplasmosis caused by large forms of *Histoplasma* could be admitted as a working hypothesis. In the same year, A. Dubois, P. G. Janssens, and P. Brutsaert described, in a white male who had lived in the Belgian Congo for the previous six years, swellings of the lymph nodes in the neck, axillary, and inguinal regions. My opinion was requested as to the presence of large ovoid cells with a thick double-contoured membrane, from 13 to 15 μ by 10 μ. Although some pathologists suggested blastomycosis, there was, I thought, no possibility of making a diagnosis.

On Sabouraud agar, at 25° C., there appeared a slightly fluffy fungus, white at first, brown later. A tendency to pleomorphic degeneration developed after many subcultures. On microscopic examination there appeared a vegetative and an aerial mycelium with spores of three types: (1) small, round microconidia 2.5 μ in diameter, growing on short sporophores along the mycelium; (2) large, smooth macroconidia; and (3) echinulated macroconidia both supported by long sporophores. The macroconidia, 15 μ in diameter, were round without exception: this was their sole difference from macroconidia in *H. capsulatum,* with which the newly isolated fungus shared so many similar features.

The culture inoculated in the testis of the guinea pig provoked a soft swelling after fifteen days. The fluid obtained by puncture of the testis at this stage showed a fair number of histiocytes invaded by parasites very similar to *H. capsulatum*. Later these fungi enlarged and, by the end of the third or fourth month, every parasite resembled in every respect those found in a patient by Dubois and his colleagues. I named the small forms first observed the *capsulatum* forms; the larger ones, the *duboisii* forms; and I coined for the fungus the name of *Histoplasma duboisii* n. sp.* From a perusal of the literature, eight possible cases of histoplasmosis caused by *H. duboisii* were found (Lecene, 1936; A. Catanei and P. Kervran, 1945; P. Kervran and R. Aretas, 1947; Friess and Delvoye, 1947; G. T. Duncan, 1947 and 1956; and G. T. Duncan, personal communications). Since then other cases have been described (G. H. Clarke, J. Walker, and R. M. Winston, 1953; W. D. Silvera and P. G. Konstam, 1954; G. Audebaud, P. Merveille, P. Laluque, and R. Depoux, 1954). Typical *duboisii* forms have been observed in each case (fifteen in all), and every one of them originated in Africa. This fact allowed Dubois and myself (1953) to raise anew the question of an African histoplasmosis caused by *H. duboisii* that existed in Africa along with the classic histoplasmosis caused by *H. capsulatum*.

Much interest was raised by my consideration of the fungus as a species, and in 1953 I wrote: "For the different reasons explained above, I thought it justifiable to create the new species *H. duboisii* (Vanbreuseghem 1952). It seems from the citations made in the beginning of this paper that this species has been discovered by other workers, but it had been generally considered to be *H. capsulatum*. Duncan was the sole author to suggest that it might be a new species. We shall learn in the future if I was right."

R. M. Crumrine and S. F. Kessel (1931) were the first to observe in the spleen of a 42-year-old man (dead of histoplasmosis) some large forms. J. Schwarz (1953) reported the presence of giant forms of *H. capsulatum* in tissue explants, and of similar forms in a caseating adrenal gland. C. H. Binford (1955) wrote that he found large extracellular forms of *H. capsulatum* in necrotic adrenal glands, in a necrotic renal medulla, and in valvular lesions. It is important to note that the same author observed in necrotic tissues some septate hyphae that A. A. Humphrey (1940) had already observed in a pulmonary cavity. L. A. Weed (1953) had also described the variations in size in parasitic morphology of *Blastomyces* and *Histoplasma*. In 1934 W. A. de Monbreun had already ob-

*The description runs as follows (Vanbreuseghem, *in* Dubois, Janssens, and Brutsaert, 1952): *H. duboisii* has two forms in the parasitic state: a small one — capsulatum form — which is similar to the only one known in *H. capsulatum* and a large ovoid form, 13 μ by 10 μ — *duboisii* form — with a thick membrane that produces a bud that separates from the mother cell by fission. In the saprophytic state one finds microconidia 2 to 3 μ in diameter along hyphae, and smooth and echinulated macroconidia with a double-contoured membrane. The diameter of these macroconidia is about 13 to 15 μ. The macroconidia are perfectly round and are supported by long sporophores (to 30 μ or more). The yeast phase of *H. duboisii* is described later.

served on blood agar slants inoculated with *H. capsulatum* some yeast cells two to three times larger than the normal yeast cells.

In a paper written on this subject in 1956, I pointed out, however, that "on the basis of the studies heretofore reported we may admit: (1) that in tissues infected by *H. capsulatum*, large forms which may reasonably be attributed to that fungus occasionally appear; (2) that similar forms may develop in tissue explants or in aged colonies of the yeast phase of *H. capsulatum* at 37° C.; (3) that *in vivo* these large forms are always extracellular. They develop in necrotic, caseating tissues which probably reproduce the conditions in which the yeast phase grows *in vitro*."

At the same time E. Drouhet and J. Schwarz (1956) published their observations on the morphological variations of what they call American and African strains of *H. capsulatum*. These authors remark that in the yeast phase small and large forms may be observed, and that, although large forms are more frequent in the African strains and small ones more frequent in the American strains, there exist strains from both origins that show nearly the same morphological features. They concluded: "The large variability of American and African strains observed in the yeast phase, the constancy of the mycelial phase with the characteristic echinulated macroconidia do not seem to justify the acceptance of a distinct species to group the strains of *Histoplasma* with large forms."

On the other hand, F. Mariat and G. Segrétain (1956), in completing the study of large forms observed in a case of histoplasmosis in a baboon by G. Courtois, G. Segrétain, F. Mariat, and J. C. Levaditi (1955), came to the conclusion that it was necessary to make a clear distinction between the small forms of the *H. capsulatum* type histoplasmosis and the large forms of the *duboisii* type histoplasmosis. They propose to consider that *H. capsulatum* contains varieties differing in secondary characteristics from the type species.

Lately G. T. Duncan (1956), reporting on the paper by Drouhet and Schwarz (1956), noticed that "The description of the large forms in the hamster and the supporting photograph illustration show them to be different from the different large tissue forms of *H. duboisii* and suggest that they may be of the same nature as the giant forms seen by Schwarz in cultures of tissue explants."

From all the facts known thus far there seem to be very satisfactory reasons for distinguishing clearly between *H. capsulatum* Monbreun 1934 and *H. duboisii* Vanbreuseghem 1952. The arguments favoring recognition of the last named as a species may be summed up as follows:

(1) All cases of histoplasmosis caused by *H. duboisii* are of African origin.

(2) The nearly complete similarity between *H. capsulatum* and *H. duboisii* in culture does not seem to work against their distinction as

species any more than a similarity *in vivo* is against a distinction of species recognized as such by their morphological features *in vitro*. Although *T. ferrugineum, M. audouini,* and *M. canis* produce in the living hair the same typical microsporic aspects, no one has opposed recognizing them as different species based on their characteristics *in vitro*.

(3) The large forms observed in a few rare cases of histoplasmosis caused by *H. capsulatum* are not similar to the *duboisii* forms of *H. duboisii*. My arguments on this subject run as follows (Vanbreuseghem, 1956):

"(a) The *duboisii* form of *H. duboisii* in man is the only recognized parasitic form of *H. duboisii*. This parasitic form is also the only one to be found in animals some weeks after inoculation.

(b) In living tissues the *duboisii* form is always intracellular. On the contrary, the so-called large forms of *H. capsulatum* are extracellular and are to be found in necrotic tissues.

(c) In the guinea pig, during the first weeks after intratesticular inoculation, there exists a form of *H. duboisii* which is very similar to the parasitic stage of *H. capsulatum:* that is why I named it the capsulatum form of *H. duboisii*. But in some weeks' time this capsulatum form disappears completely and the *duboisii* form is the only one to be found.

(d) A comparison between the *duboisii* form of *H. duboisii* and the so-called large forms of *H. capsulatum* shows readily that these cells have more differences than similarities. Of course, both of them are large cells, but whereas the *duboisii* forms are nearly always ovoid, with a very regular double-contoured membrane, the large forms of *H. capsulatum* are quite irregular and are rarely or never ovoid (see Schwarz, 1953, and Binford, 1955).

(e) In necrotic tissues of animals infected by *H. duboisii,* some extracellular forms develop. They are larger than the intracellular forms and much larger than the extracellular large forms of *H. capsulatum*. Both parasites behave similarly in similar conditions.

(f) In cultures of *H. duboisii* maintained at 37° C. to obtain the yeast phase, large forms develop, but they are always larger than the large forms produced by *H. capsulatum* under the same conditions."

(4) A. Montemartini and O. Ciferri (1956) have demonstrated that the amino-acid requirements of *H. duboisii* and those of *H. capsulatum* are different, and they conclude: "From the assimilation tests with amino acids and other nitrogenous sources it should be concluded that the two species of *Histoplasma* possess some autonomy although they have a common relation."

(5) J. Coudert and M. Coly (1956), using the collodion particle agglutination test, come to the conclusion that "by this reaction American histoplasmosis may be distinguished from African histoplasmosis caused by *H. duboisii,* although an antigen seems to be common to both affections and another is found in African histoplasmosis."

(6) While the tissular reactions produced by *H. capsulatum* are of the histiocytic type, *H. duboisii* produces mainly a reaction of the Langhans type. This fact has been observed experimentally by Dubois and Vanbreuseghem (1956) and in a recent human case by Vandepitte and his colleagues.

It would probably be easy to criticize each of the arguments proposed in favor of considering *H. duboisii* as a species but, on the whole, these contentions have, in my opinion, much value favoring the acceptance of *H. duboisii* Vanbreuseghem 1952.

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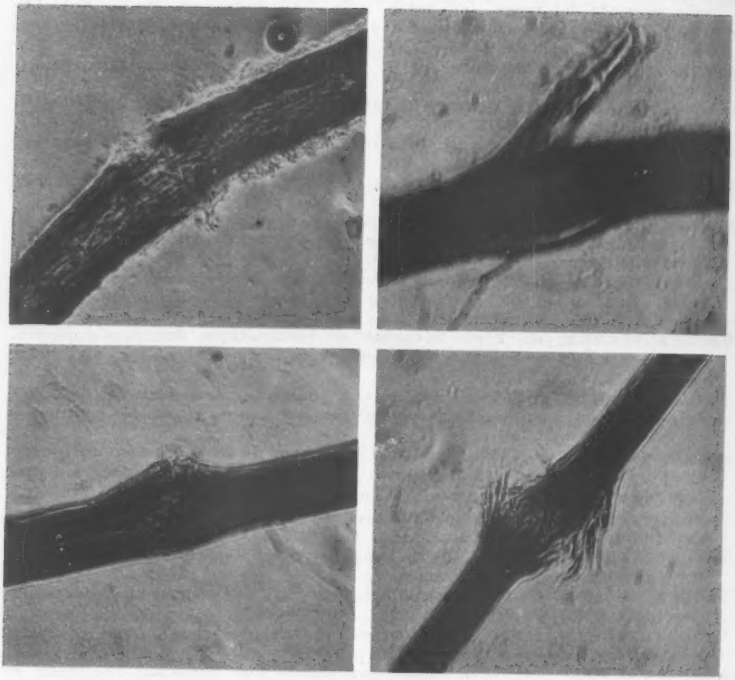

FIGURE 1. Typical lesions of the hair in kwashiorkor (malignant malnutrition).

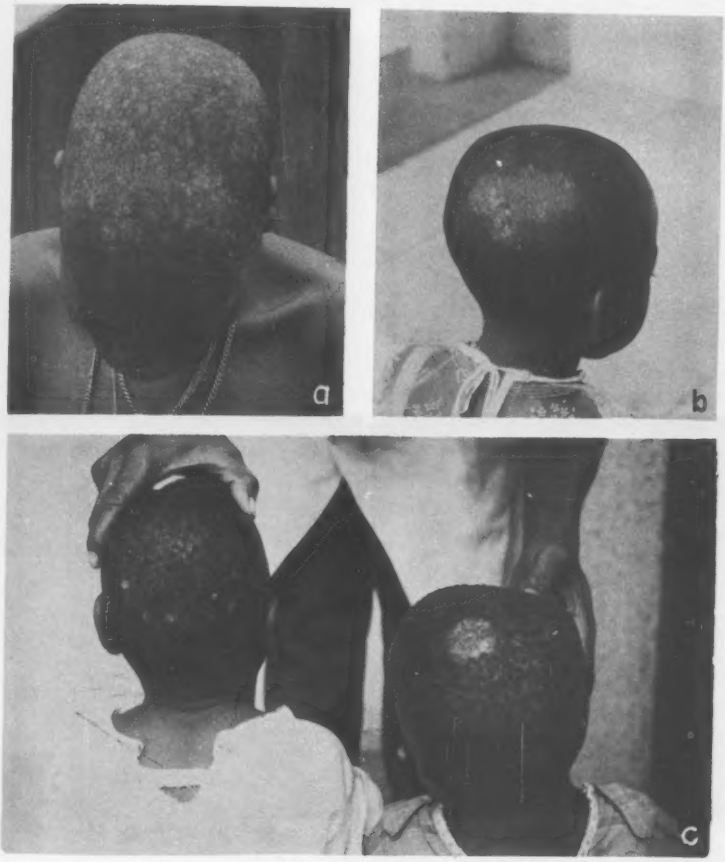

FIGURE 2. (a) Tinea capitis by *Trichophyton violaceum* in a 27-year-old woman in Ruanda-Urundi. (b) Tinea capitis by *Trichophyton (langeronia) soudanense* in a 3-year-old girl in Léopoldville. (c) Tinea capitis by *Microsporum (Sabouraudites) langeroni* in two sisters 5 to 7 years old in Léopoldville. In one of them the lesion has a microsporic character; in the second (the larger child) it resembles a trichophytia.

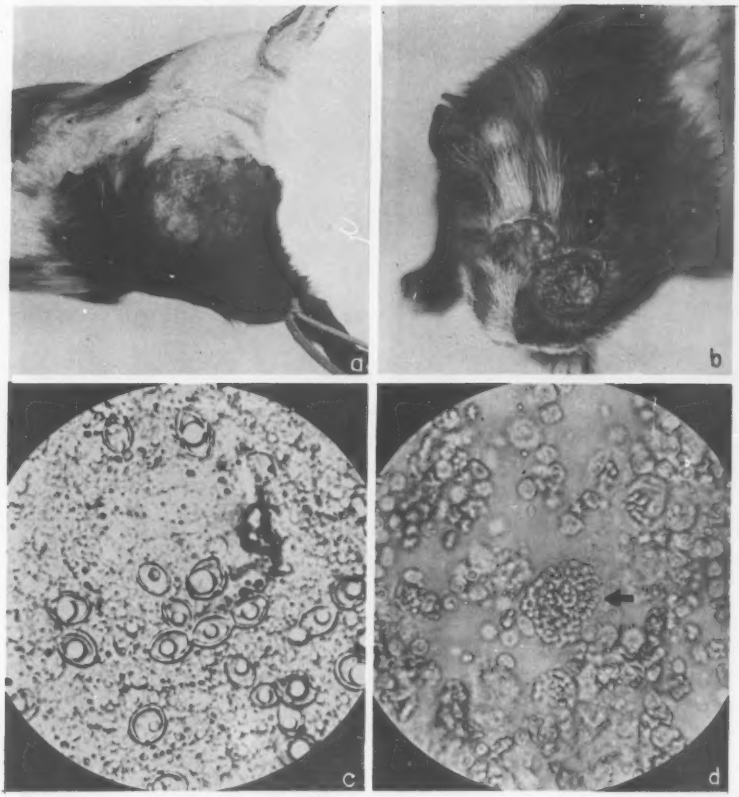

FIGURE 3. (a) Guinea pig inoculated 10 months previously with pus from a case of African histoplasmosis *(Histoplasma duboisii)* in the right testicle. (b) Guinea pig inoculated 9 months previously with pus from a case of African histoplasmosis *(H. duboisii)* in the right testicle. Typical hyperkeratotic lesion of the face. (c) *Histoplasma duboisii* in the pus of a lymph node from African histoplasmosis. Typical *duboisii* forms. ×250. (d) The *capsulatum* form of *H. duboisii* in the testis of a guinea pig 15 days after intratesticular inoculation of a culture of *H. duboisii*. ×500.

SECTION OF GEOLOGY AND MINERALOGY

GEOLOGY AND RELATED ENGINEERING PROBLEMS AT THULE, GREENLAND*

By Helgi Johnson

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While employed in the Military Geology Branch of the United States Geological Survey, Washington, D.C., Alan H. Nicol and I were given the opportunity to go to Thule, in northwestern Greenland, to act as observers when that air force base was under construction. The code name of the base in October 1951 was Operation Blue Jay. The trip was sponsored by the United States Corps of Engineers, and this organization, in over-all command of the project, made available all engineering reports and data on the site. Of particular interest were reconnaissance reports on the geology by Joseph M. Trefethen; *Engineering Characteristics of Soils in the Thule Area* by Robert Frost; *The Ground Water Resources,* a report compiled by the United States Air Force; and data on earth temperatures by the Northeast District, United States Corps of Engineers. Because all of this information was highly classified at the time, no direct quotations or exact figures based upon the reports are cited in this paper. I recall with pleasure the many courtesies extended to me by the district engineer in charge of construction and his staff, by the staff of the architect engineers, Metcalf and Eddy, and by Alfred Hopkins and Associates, the construction contractors of the project.

Location and Access

Blue Jay or, as it is now known, Thule Air Force Base, is located in an ice-free, east-west trending valley about three miles south of the little Danish trading post of Thule in northwestern Greenland. Except for a summer station at Etah, about 125 miles to the north, this was the northernmost human habitation in Greenland. Thule, a locality well known to early and recent explorers, is open to navigation for two months of each year. Prior to our undertaking of major construction, the site had been chosen as a weather station jointly operated by Denmark and the United States, and it had a short unsurfaced runway on which aircraft could land

*This paper, illustrated with lantern slides, was presented at a meeting of the Section on December 3, 1956.

The writer takes full responsibility for the contents of this report and it does not necessarily follow that the views expressed are officially those of the United States Corps of Engineers or the United States Geological Survey. The author further recognizes the fact that much research has gone on since the time of his visit. He has not had access to the resulting information.

Publication of this paper has been authorized by the United States Geological Survey and the United States Corps of Engineers, Washington, D.C.

to bring in supplies and change of personnel. In 1950, it was decided to expand this facility to a modern air force base, and preparations and initial phases of construction soon followed. More recently, Scandinavian Airlines, having in mind a polar route for commercial traffic on their Orient run, have made experimental landings there.

The valley, approximately twelve miles long and two miles wide, opens into North Star Bay, on the west. The Greenland Ice Cap forms the eastern terminus; and North and South Mountains, two hogback ridges, border it in these respective directions. The valley floor, a sloping one, rises from a few feet above sea level to a little over a thousand feet at the foot of the ice cap.

Climate

Technically, the climate of the Thule region and, in fact, of most of Greenland, is "ice cap climate." To be trite, this means very cold and very dry; there are no frost-free days (twenty-four hour periods) and the annual precipitation is less than three inches, mostly as snow. Winds of high velocity are common (up to 100 knots) and are from the direction of the ice cap in most instances. The thaw periods begin in June and last through August; the mildest weather occurs in July and early August.

Surficial Geology

Glacial moraine (till) partly fills the valley and it varies in thickness from two or three feet to more than 100 feet. The surface is a pitted plain having two main drainage channels cut into it to a depth of only a few feet. Several small lakes occupy kettle holes, and at least two of these lakes are sufficiently deep so that they do not freeze to the bottom during winter. Microrelief on the moraine seldom exceeds ten feet, but some bedrock ledges that stand above the surface rise as much as fifty feet. Since the recession of the glacier ice, there has been considerable resorting and redistribution of the unconsolidated surface materials. Low areas are frequently partly filled by wind-blown silt and fine-grained sand (loess); higher areas are developed into desert pavements of varnished boulders and cobbles, and all of the fines have been blown away. Everywhere there is developed the characteristic patterned ground so typical of permafrost areas that are subjected to short thaw seasons. Where bedrock is exposed, there has been considerable, although unequal, development of felsenmeer, especially on sandstone surfaces; less development has occurred on sloping diorite and dolomite surfaces. Spalling and talus accumulations are common phenomena on the latter two surfaces.

Considerable tundra vegetation was present in the more sheltered parts of the valley; hardy alpines are even now invading the freshly accumulated moraine at the foot of the ice cap, and lichens coat the bare rock surfaces of even the highest hills.

Bedrock Geology

Igneous, sedimentary, and metamorphic rocks are found in the Thule area. The oldest of these are metasedimentary rocks of probable Pre-Cambrian age. They form a basement complex for later unmetamorphosed sedimentary rocks and crop out along the southeastern shore of Wolstenholm Fjord to the edge of, and presumably underneath, the ice cap. The dominant lithology of the metasediments is garnetiferous gneiss and schist, with one small area of skarn. However, granite gneiss (quartz-oligoclasebiotite gneiss) is a very common constituent in the present terminal moraine, and it must form a considerable part of the same terrain. It is also interesting to note that a single cobble consisting of hematite similar to that found in Huronian rocks was chipped from an overhang at the base of the ice cap, but such slim evidence scarcely warrants an assumption that less metamorphosed, younger Pre-Cambrian sedimentary basins exist under the ice.

Overlying the basement rocks is a thick sequence of unmetamorphosed sedimentary rocks containing several concordant sills and lenses of intruded mafic igneous rocks. At least two nearly vertical dikes also intrude the sediments south of the valley.

The stratigraphic section begins with a thick sequence of arkosic sandstones that unconformably overlie the basement rocks. Many observers have referred these to the Thule sandstone of supposed late Pre-Cambrian or Cambrian age; except for a general similarity of surface coloring, it is impossible to correlate them accurately with the measured type section of the Thule that crops out in the Etah area, 125 miles north of Wolstenholm Fjord. Without apparent unconformity, the basal sandstone seems to grade upward into shaly sandstones, black carbonaceous shales, and lighter sandstone, weathering to a yellow to buff color. Although these rocks have yielded no identifiable fossils, I have observed leaflike impressions containing carbon films on many slabs of the shale.

Overlying the light-colored sandstone are concordant calcareous siltstones containing frequent lenses and ribbonlike beds of selenite. These, in turn, grade upward into impure massively bedded vuggy dolomites and limestone with recurring thin beds of gypsum and anhydrite. The only fossils found in this thick carbonate sequence are two unidentified algal heads, each about a foot in diameter. Time did not allow either a careful measurement of the section or a serious search for fossils, but the presence of so much gypsum in the carbonates and carbon and possible leaf impressions in the shale makes one suspect that we are dealing with rocks of the late Paleozoic or Mesozoic age rather than with Pre-Cambrian or Cambrian strata.

Igneous Rocks

Numerous sills sill-like lenses, and at least two vertical dikes are

intrusive into the sedimentary rock sequence. The sills seem to be confined to the basal half of the succession and crop out on both shores of Wolstenholm Fjord, and also as intersects in diamond-drill boreholes in the valley. Dundas Mountain is capped by such a sill with the sedimentary cover removed by erosion. The extension of the Dundas Mountain sill also forms the dip slope of North Mountain. In composition the sill rocks are all lumped together as diabase, because lath-shaped crystals of plagioclase feldspar is a prominent mineral component of the rock. Epidote coats joint and fracture planes and gives the rock a greenish color when broken.

The dike rocks are also mafic and probably are similar in composition to the sill rocks. There is little doubt that both sill and dike rocks are Cenozoic in age, probably being related to the plateau basalts so prevalent in other parts of Greenland.

Structure

All the rocks younger than the metasedimentary rocks occur in a gently folded, east-west trending syncline with the steeper dips on the north flank and gentler dips to the south. Normal faults with strikes parallel to the axis of the syncline are visible, but their number and the degree of offset were not determined at the time of our visit.

Engineering Aspects

Many engineering problems in the area have direct geologic involvements, and some relate directly to the presence of permafrost, found everywhere in North Greenland. I do not intend to discuss details of construction, but I may say that all of the buildings are prefabricated, aluminum-clad, heavily insulated and laminated, module-planned units. All but the hangars and power plant buildings stand on stilts with extra heavy insulation in the floor to prevent or to lessen freeze-thaw cycles through heat transfer in the cold season. The site for each building was prepared by placing a gravel "pad" to an established grade. The obvious reason for this practice is to improve drainage and to prevent unequal settling in the summer months. How successful this designing is can be proved only after a number of years for, even without such precautions, buildings with footings in permanently frozen soils are slow to settle.

Because of strong winds these buildings are constructed with outside sills at the bottom of the walls, with extra heavy concrete slabs placed on them for anchoring purposes.

The heavy service buildings, the hangars, the runways, and stands must be constructed at ground level in conventional fashion. The thickness of unconsolidated material (moraine) and the fact that all but a few feet of it near the top, constituting the active layer, is permanently

frozen, precluded the normal driving of piling or sand drains to stabilize the subgrade and base course of the foundations, the floors, and pavements. Furthermore, in the process of excavating the active layer, many of the attendant phenomena of permafrost, such as ice wedges and large ice lenses, were met. It became necessary to remove not only the active layer, but all ice lenses wherever found, and to replace all fine material below the active layer level to considerable depth. The material so removed was replaced by coarse gravel and compacted without fines. Drains were installed at three critical levels corresponding with thaw penetration of two months' duration, and surface drainage radiating from these structures in all directions was established.

Based upon Alaskan experiences, it was thought that ground water in considerable quantity might be found in the sandstones at depths below the permafrost levels. Consequently, a vigorous drilling program was pursued at locations that looked most promising. It was considered that earth temperatures might be warmer underneath the bottom of the bay as well as underneath the ice. Therefore, the first holes were located as near to the shore and ice cap as convenient. One hole was located on an ice boil where upward welling of water formed a large dome of ice. None of the drilling succeeded in penetrating through frozen ground and rock that extend to depths of more than a thousand feet. The temperature at that depth, as measured on a constant recording thermometer, was 28° F. Drilling was stopped and hope abandoned for a source of ground water.

The lakes were also considered as a water supply, but the lake water, though potable, was too hard (several hundred parts per million) to be used for generating steam. Also, it was estimated that the volume of water in the lakes that did not freeze would not suffice for our needs. Careful consideration was also given to the possibility of obtaining water from melting glacial ice, but this was considered impracticable because of the distance to the cap and because of the high consumption of fuel needed to melt the ice. Therefore, the present water supply is distilled sea water or it is obtained from lakes.

Aggregates of unconsolidated and crushed bedrock were used in various phases of construction. The unconsolidated material was fairly well-sorted gravel in a delta that had been elevated by changes in eustatic levels. The gravel consisted largely of pebbles of gneiss and sandstone, but it also contained shale, which rendered it useless for concrete. However, it proved to be an excellent and cheap source of replacement material for unsuitable subgrades. Diabase from North Mountain and dolomite from South Mountain were both quarried and used where specifications demanded good, sound stone.

Because it was planned to use the ice cap as a staging area and, more important, as an emergency-landing or crash-landing area with semi-permanent air rescue stations at specified locations, this vast and deso-

late region has been the subject of important engineering and glacial studies since the early phase of base construction. The chief responsibility for such research rests with the Snow, Ice, and Permafrost Research Establishment under the direction of the United States Corps of Engineers, working with the cooperation of the United States Air Force.

I had very limited experience with the ice cap: a single traverse across the front and one short traverse on foot for a distance of about three miles up the slope. The front, except where the ice disgorges through valleys between solid rock walls where all of the characteristics of Alpine glaciation are duplicated, is perpendicular to an overhanging cliff of stratified ice up to three hundred feet in height. Two exceptions were noted where tongues or lobes of ice had a gently sloping surface. Tracked vehicles such as weasels and tractors could move up the slope to a distance of twenty to twenty-five miles from the front. Here a crevassed zone prevented further progress but, since 1951, other routes have been surveyed and are usable by traffic.

In winter there is sufficient snowfall and drifting to form sastrugi on the surface and snow ramps extending from the top to bottom of the cliffs. These ridges generally melt away in summer, leaving a gleaming ice pavement. Melt waters form a consequent stream pattern on the natural slope with valleys six to seven feet deep and twenty to thirty feet wide.

The ice front shows a great variety of features, the most notable being the stratification of layered ice. Some layers are blue-green, some are crystalline, and some display included water bubbles. Generally the layers show upward bending on the face, to the extent that morainic materials have been carried from the bottom to the top. Other flow features on a lesser scale are evident. Crenulations and some folding and thrust faulting are not uncommon features, and many healed fractures are immediately apparent.

SECTION OF MATHEMATICS AND ENGINEERING

THE RECRYSTALLIZATION OF SOME PROCESSED METALS UNDER VARIABLE STRESS*

By John N. Kenyon

Fatigue of Materials Laboratory, Princeton, N. J.

Introduction. The theory of fatigue phenomena entertained a century ago was that a metal crystallized and failed under cyclical stress. About the turn of the century, the crystallization concept was abandoned with the advent of metallography, and the "phenomenon of the spreading crack" was substituted. So sweeping was the latter concept that it virtually became a definition, and consideration of *any* crystalline change was practically ruled out. The findings reported herewith prove conclusively that crystalline changes occur under variable stress and that they may well account for service failures otherwise unexplainable. Certain mathematical misconceptions will be discussed.

Philosophical concepts of research approach. My first concept of research was that of a science; today it appears to be also a philosophy. While some of the opinions expressed herewith may seem extreme, all would seem to have important bearing on the philosophy of research approach.

The spokesman for a research institute was summarizing the advances made in the radio field when a boy challenged one conclusion. The speaker rebuked him: "Young man, there was a time when an untrained person could accomplish in science; *that day has long since passed.* Research must now be carried on with a large organization, expensive equipment and ample money." This incident occurred in 1923. (Two years previously, Edwin H. Armstrong, with a few amateur associates, had made history by spanning the Atlantic by short-wave radio.)

Arthur H. Compton entered a classroom at Columbia University, New York, N. Y., appearing somewhat nettled, and remarked: "I feel like booting myself all over the campus." Compton explained that he once had had a research lead, but that the idea had been crushed. Another scientist had now carried out the work and the findings had just been published. This was in 1933.

C. E. K. Mees, of the Eastman Kodak Co., Rochester, N. Y., remarked to a visiting group of bankers and industrialists: "The best person to decide what research shall be done is the man who is doing the research. The next best is the head of the department. After that you leave the

*This paper, illustrated with lantern slides, was presented at a meeting of the Section on February 15, 1957.

field of best persons and meet increasingly worse groups. The first is the director, who is probably wrong more than half the time. Then comes the committee, which is wrong most of the time. Finally, there is the committee of company vice-presidents, which is wrong all of the time." Mees was then vice-president and research director of his company.[1]

Nolan D. C. Lewis, of the College of Physicians and Surgeons, Columbia University, said, "The watertight compartment attitude of many scientific groups in universities and elsewhere is not conducive to progress along a broad front. Every advance in science is made at the expense of someone's reputation as an authority. We should be *stimulated by authority,* but not *paralyzed;* should *admire,* but not *worship.*"[2]

Edwin H. Armstrong in *Mathematical Theory vs. Physical Concept* stated that frequency modulation was proved workable "...by proceeding in exactly the opposite direction that mathematical theory had demonstrated one ought to go."[3]

Irving Langmuir intimated in a radio address that he won the Nobel Prize by departing from the assigned problem. He further stated: "...great harm might come from any attempt at regimentation based on the belief that basic research can be wholly directed from the top. The directors of laboratories are not supermen who can foresee new knowledge before it exists. Only a small part of scientific progress has resulted from a planned search for specific objectives. A much more important part has been made possible by the freedom of the scientist to follow his own curiosity in search for truth."[4]

In summation of the sundry points of view expressed above, it would appear that blind worship of authority is most detrimental to creative scholarship. This "authority" may be the directorship, or it may be blind worship of mathematical expression.

Statistical evaluation of data versus sensitivity in testing technique. Too often, the young research worker feels that, to be scientific, he must speak in the language of symbolism. He builds up a mathematical relation based on assumptions not justified by the meager data and may obtain a misleading answer. A mathematical solution thus derived is no different from a faulty setting on a slide rule; only an incorrect answer is forthcoming. Again, this research worker may start with a mathematical concept and, by way of verification, carries out a few tests by some available standardized method. He obtains wide scatter in data, employs statistical rules to eliminate the isolated points, and confirms his theory in the qualitative sense. It is the experience of the writer that wide scatter in data is usually due to lack of sensitivity in testing technique; that with proper refinement these data can be resolved into symmetrical graphs. Finally, when scatter does occur in spite of every refinement in sensitivity of technique, these scatter points may have important implications.

Illustration. In an American Society for Testing Materials (ASTM) pub-

lication of 1945, the writer advanced the theory that the speeding pneumatic tire may build up a "standing sinusoidal wave" and would more likely fail at the top than at the point of contact with the pavement. In a subsequent ordnance study,[5] the following dynamic theory was propounded: if the pneumatic tire were suspended in space as a free body and made to assume its period of natural frequency, the tire would burst into fragments. The service tire is subjected to mechanical constraints (pavement contact and wheel-axle assembly); as a result, it will undergo a series of critical vibrations that will approach (though never attain) the natural frequency period.

FIGURE 1 shows the dynamic behavior of an ordnance tire wherein instantaneous strains (stretch) are plotted against disturbing forces expressed in pulsations per minute. These data were obtained under difficult testing conditions, using a tire of heterogeneous construction under disturbing forces of a dynamic nature. Nevertheless, the strain-frequency curves obtained were parabolic and without sufficient scatter to justify the employment of band. In the earlier stages of the dynamic study made of this tire there was wide scatter in points, and the plotted data were of virtual scatter band. Had the investigator employed statistical methods to rule out isolated points, the high critical surge shown in FIGURE 1 might have been missed entirely. These maxima occurred at regular intervals for frequencies up to (and above) 12,000 per min. The symmetry of the curve is explainable by the high sensitivity of the testing technique.

Brief history of fatigue phenomena. The first scientific meeting dealing exclusively with fatigue phenomena was held in Birmingham, England, in 1848. The crystalline theory of fatigue failure was generally accepted. This opinion prevailed until the turn of the century; then, with the advent of metallography, it was arbitrarily dropped. A new concept, "the phenomenon of the spreading crack," was substituted and almost became a definition. The following statement appeared in an ASTM publication in 1950: "The 'crystallization theory' of a century ago has been proved unsound by metallographic studies."[6] The author of this assertion evidently spoke for the current school of thought and considered only data obtained on the ferrous metals. No exception can be taken to this statement, since the old concept of crystallization had assumed that the metal hardened under variable stress, lost ductility, and assumed brittle cast-ironlike properties. However, the obvious discrepancy in the concept is that cyclical stresses (fatigue) are usually below the yield point and do not have a work-hardening effect. Furthermore, little thought appears to have been given to the possibility that a processed metal might actually soften.

"Recrystallization" of some hard-drawn metals under variable stress. I became interested in the fatigue behaviors of the nonferrous metals at the instigation of W. H. Bassett, Sr., of the Anaconda Company, Hastings-

on-Hudson, N. Y., who had expressed the hope that more consideration be given to these materials. At the time I fortunately had developed two fatigue-testing techniques whereby a rod (or wire) could be tested in long lengths. The standard fatigue specimen confines failure to the center of length.

It was well known at the time that certain processed nonferrous metals had variable fatigue strengths. In fact, heats of copper might vary considerably in the fatigue value when they otherwise met with the standard specifications for chemical and static physical properties. One heat of hard-drawn copper encountered was so abnormally low in fatigue strength that the wire specimens were subjected to microscopic examination. It was found that a new crystalline phase had appeared; large "islands" of soft copper were discernible in the hard-drawn matrix. There was an apparent tendency for the soft nuclei to absorb the hard copper under the propagation of cyclical stress. The phenomenon was discovered because it appeared at every point at which the wire specimen was cut. That the softening effect had been missed by previous investigators is understandable. They had employed the standard fatigue specimen (that confines failure to center of length) and had probably ruled out scattered fatigue breaks by statistical method.

After publishing an ASTM paper in 1950,[7,8] I continued the study and have formulated the following theory:

All processed *pure* metals are in metastable condition and would ultimately revert to the soft virgin state. This reversion might be vastly accelerated under cyclical stress if the metal were at a condition of critical temperature. In processed alloyed metals, two phenomena might occur: (1) a trend of the hard-drawn properties to soften, and (2) a furthering of the precipitation hardening. The integrated effect of the two should be evident in fatigue data obtained at some higher temperature.

While it is well demonstrated that a hard-drawn copper may revert to soft metal at room conditions, the phenomenon is not so pronounced in other metals. That the effect does occur in hard-drawn aluminum is revealed by the X-ray pattern and by a lowered fatigue strength when the tests are carried out at 100° C. or more. It is not revealed in the microscope, probably because of the difficulty in etching aluminum (FIGURE 6).

Evaluation of data. The data herein reported are contained in FIGURES 1 to 6, with a brief summation in captions.

Tentative conclusions. I have formulated the theory that all processed pure metals are in metastable condition and might ultimately revert to the soft virgin state, and that this reversion may be much accelerated under cyclical stress. Furthermore, I believe that all pure metals in a hard-drawn condition will so revert at some critical temperature condition. The phenomenon occurs in copper at room temperature and is discernible under the microscope. The X ray and lowered fatigue strength indicate that it also occurs in hard-drawn aluminum at 100° C. or more.

Prediction is made that the reversion may occur even in hard-drawn iron materials at some critical temperature. Finally, just what is the point of incipient beginning where soft metal forms and tends to absorb the hard-drawn matrix? Is it possible that some contained impurity acts as a catalyzing agent that precipitates recrystallization and the advent of the new solid phase? More research will be needed to answer these questions.

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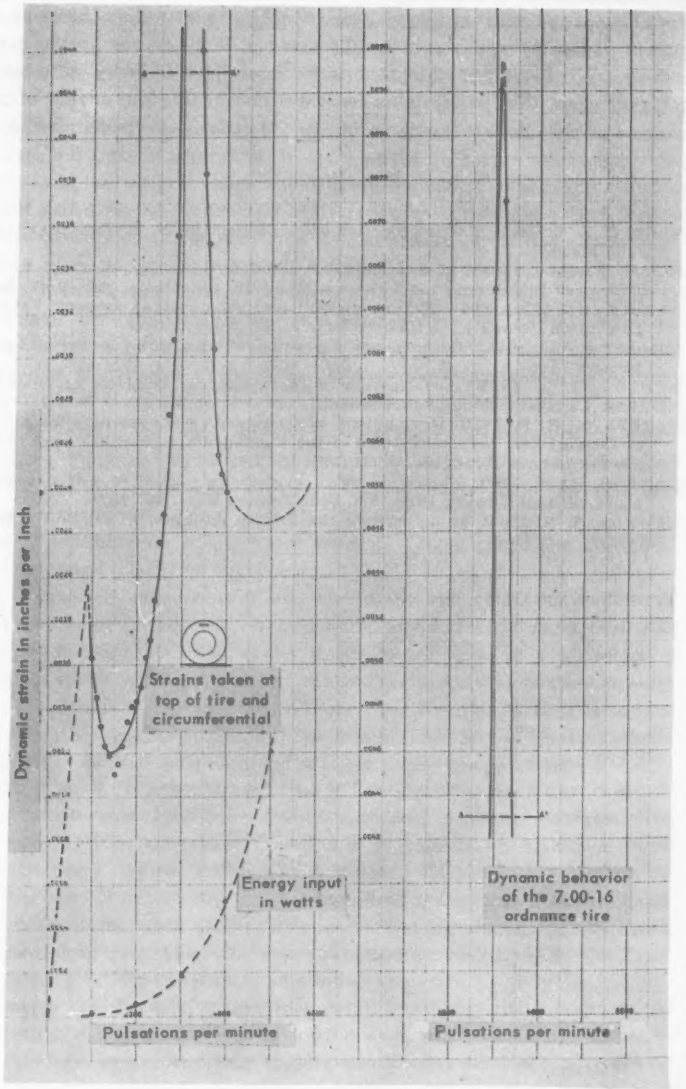
Dynamic strain in inches per inch
Strains taken at top of tire and circumferential
Energy input in watts
Pulsations per minute
Dynamic behavior of the 7.00-16 ordnance tire
Pulsations per minute

FIGURE 1. The dynamic strain frequency curve obtained on an ordnance tire. Note that the high dynamic surge obtaining at 3100 pulsations per min. (PPM) reoccurred at regular intervals up to 12,000 PPM. Note also that the high dynamic surge is independent of the energy input in the tire as expressed in watts.

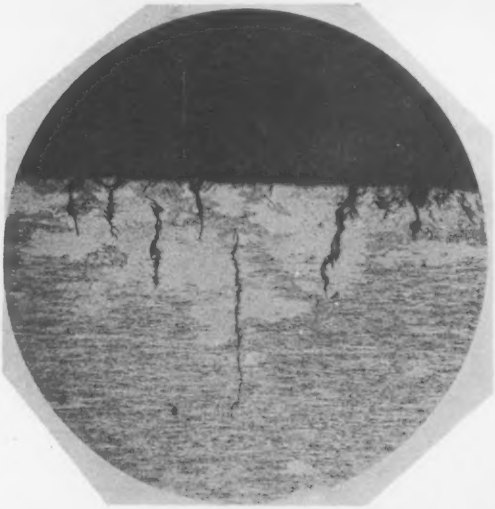

FIGURE 2. Longitudinal view of hard-drawn copper wire (stock material). ×75.

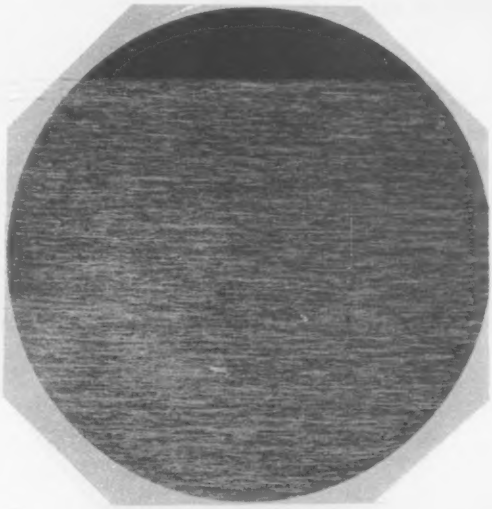

FIGURE 3. The hard-drawn copper wire after 23.9×10^6 stress reversals at 13,700 psi at room conditions. Only the outer fibers of the specimen were subjected to the maximum fatigue stress and soft copper formation was confined to this periphery. ×75.

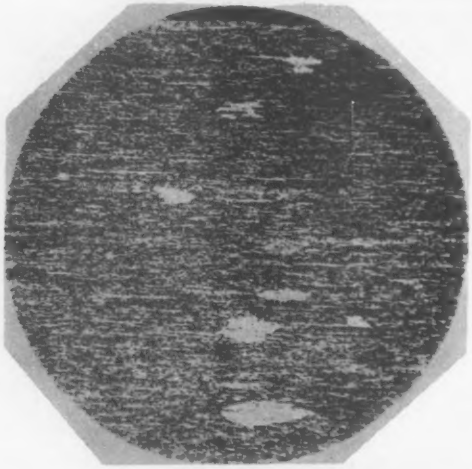

FIGURE 4. "Lake-like" areas of soft copper appearing in the hard-drawn wire after subjection to the pulsating-tension test. Here the entire cross section of the specimen and the outer fibers were subjected to the same fatigue stress (longitudinal section). ×75.

FIGURE 5. A "lake-like" area of soft copper shown under higher magnification (see FIGURE 4). Higher magnification did not break down the soft copper into its crystalline components, indicating: a grain structure infinitely fine; the structure that of single crystal and/or structure cubical, one-direction with boundary demarcations not discernible. ×500.

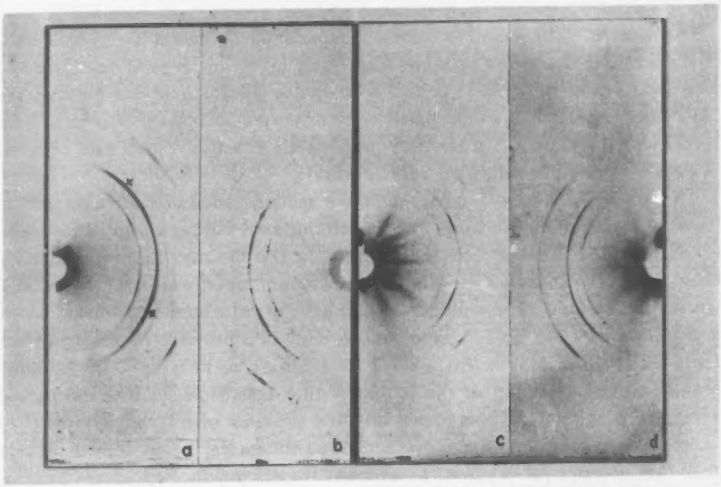

FIGURE 6. X-ray patterns on hard-drawn copper and aluminum wire.

(a) Copper wire before the fatigue test. The continuous band, heavier at the crosses (x's) indicates a fine structure with preferred orientation (see FIGURE 2).

(b) Copper wire after the fatigue test. The spotty or discontinuous band indicates the advent of a new crystalline phase in soft copper (see FIGURE 3).

(c) Aluminum wire before the fatigue test. Note the continuous band indicating preferred orientation as in *a*.

(d) Aluminum wire after the fatigue test. The spotty or discontinuous band indicates the advent of large soft crystals. This effect was obtained at 100° C., but was not distinguishable under the microscope, possibly due to lack of an effective etching technique.

SECTION OF PSYCHOLOGY

RELATIONS BETWEEN AUDITORY PSYCHOPHYSICS AND AUDITORY ELECTROPHYSIOLOGY*

By Walter A. Rosenblith

Department of Electrical Engineering, Research Laboratory of Electronics, Massachusetts Institute of Technology, Cambridge, Mass.

The title of this paper is perhaps unnecessarily restrictive. Two sets of data exist: one set describes the ways in which organisms discriminate auditory stimuli; the other describes the anatomical and physiological characteristics of those parts of the nervous system that seem to be involved with acoustic stimuli. I propose to examine these two classes of data for possible relations between them.

To this day, the most comprehensive modern summary of facts in the field of audition is *Hearing: Its Psychology and Physiology*, the monograph by S. S. Stevens and H. Davis.[1] In the preface the authors state their credo: "Theories flourish on a certain sparseness of facts and wither in the face of abundance. When all the relations are known, alternative theories are no longer possible and, if a present inventory of the facts of audition leaves little room for theories of hearing – in the nineteenth century meaning of the phrase – that situation must be accounted a sign of progress." Their views on "the problem of psychophysiological relations in hearing" are, however, less sanguine. Here they conclude that "an attempt, at present, to account for all psychological discriminations in terms of physiological processes is obviously premature. . ."

Nearly two decades have passed during which our knowledge of hearing and of the neurophysiology and neuroanatomy of the auditory system has advanced rapidly. The *Bibliography on Hearing*[2] bears eloquent testimony to the rate at which facts have accumulated in this field. There have been many excellent discussions of important auditory problems**, and yet there has not appeared on the contemporary scene a single author or authors willing to undertake the exhilarating reappraisal that seems to be required. What follows makes no pretense at being (even under the most liberal credit terms) a down payment on this job; rather, it partakes of the hors d'oeuvre designed to whet the appetite for a meal the chef of

*This paper was presented at a meeting of the Section on October 15, 1956.
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**Since 1938 (and the list is obviously incomplete) there have appeared books by H. Fletcher, E. G. Wever, Wever and M. Lawrence, I. J. Hirsh, O. F. Ranke, R. Feldtkeller and E. Zwicker, and others; chapters in E. G. Boring's books, in the *Handbook of Experimental Psychology* (S. S. Stevens, Ed.), in R. S. Woodworth and H. Schlosberg, in C. E. Osgood, in C. T. Morgan and E. Stellar, in F. Bremer, in H. Piéron, in F. A. Geldard, in J. F. Fulton's *Textbook of Physiology*, in the *Annual Review of Psychology* and *Annual Review of Physiology*; and in a review article by R. Galambos.[3]

which has not yet been chosen.

After this *caveat emptor* we are almost ready to take a bird's-eye view of the salient psychophysical and neurophysiological features of the contemporary scene in audition. Our examination may be rendered more perceptive if we first list those major developments in instrumentation and technique that have "shaped" the landscape.

Advances in Instrumentation and Technique

I should like to begin by rendering homage to G. von Békésy who, for almost three decades, has contributed outstandingly to many aspects of the field. Békésy's development and mastery of a large repertory of high-precision techniques make him a disciplined interdisciplinary team unto himself. It is to the Békésy "team" that we owe much of our understanding of the functioning of the real cochlea (in contrast to the way the cochlea was supposed to behave according to Helmholtz's theory).

Békésy and the rest of us have benefited from the extraordinary progress in electronic apparatus (and associated programming and recording equipment) that characterized the postwar period. Tailor-made acoustic stimuli of considerable complexity are now available to the experimenter. He can present to his subject various types of speech sounds (these sounds may be either natural or synthetic, intact or systematically obscured and mutilated), tone pips, filtered clicks, interrupted white noise, or all of these woven into intricate sequences of precisely timed acoustic events*.

Contemporary electrophysiologists make use of differential amplifiers, cathode followers, and even of frequency modulation in the recording of electrical activity onto magnetic tape. Problems of band width and triggering circuits have been solved in connection with radar and nuclear technology. The business end of electrodes has shrunk in size to a point where today's ultramicroelectrode permits intracellular recording. Novel physiological techniques (strychnine, immobilized unanesthetized preparations, and recording from implanted electrodes in unrestrained animals) have enormously broadened the spectrum of electrical data available. Both space and competence are lacking to do more than to state that there have been important recent developments in the histology, microbiochemistry, and electronmicroscopy of the ear.

Methods for controlling animal behavior have become considerably more sophisticated: the use of standardized recording techniques lends itself to more quantitative descriptions of response behavior. Blough[4] has shown that techniques developed in connection with learning experiments can help us obtain psychophysical data from pigeons. Considerations of

*Contrast this state of affairs with the decade that preceded publication of the Stevens-Davis monograph. Then the pure-tone oscillator, the electronic amplifier, and the oscilloscope dominated the scene, requiring tender care from researchers who desired to exploit them successfully.

experimental design, together with the rise of government laboratories, have led to a greater variety of experimental subjects, instead of the traditional ones – the highly trained laboratory inmate and his brother under the skin, the college sophomore.

Advances in Auditory Physiology

After this sketchy review of instrumentation problems I shall enumerate what to me are the most significant advances in the physiology and, in particular, in the neurophysiology of audition.

(1) Our knowledge of the gross mechanical properties of the cochlea and of the cochlear vibration patterns for pure tones is reasonably well established.

(2) We possess a fairly precise description of the various electrical events in the cochlea, although we still lack a definitive picture of the role of these potentials in the initiation of activity in the auditory nerve.[5] In spite of these important additions to our knowledge of the cochlea, we still cannot give a reasonable account of the over-all physiological mechanisms that underlie even the most familiar psychological phenomena of hearing. This state of affairs, coupled to the general development of electroneurophysiology, has led to an intensified search for relevant neural mechanisms in order to supplement the intricate cochlear mechanisms that have proved insufficient in accounting for the behavior of the total organism.

(3) We have started to compile a systematic catalogue of input-output relations between neuroelectric responses from various stations of the afferent auditory system and acoustic stimuli, the spectral or temporal characteristics of which are particularly simple, that is, pure tones and clicks. The analysis of the electrical responses recorded by either gross or microelectrodes has made use of such concepts as *response areas*, *tonotopic organization*, and *intensity* and *recovery functions*.

(4) Modern neuroanatomical techniques have helped to discover – or rediscover – many important features of the classical ascending auditory pathway. The use of various types of unanesthetized preparations has led to findings of great functional significance: there exists, in addition to the classical pathway, an as yet undifferentiated ascending reticular pathway.[6] The two parallel ascending systems are in some sense balanced by a multiplicity of descending pathways. Both the reticular ascending pathway and the so-called "centrifugal" pathways are responsive, not only to acoustic stimuli, but also to other sensory stimuli* and perhaps even to changes in the *milieu intérieur*.

(5) Carefully controlled studies on animals with lesions in the auditory system have emphasized the extent to which this system is flexible (or should one say redundant), especially when the tasks put before the

*We may thus be able to study mechanisms that underlie intersensory effects.

animal are simple discriminations.

Auditory Psychophysics

Six areas have been among the most active:

(1) The predominant theme of the past decade has been man's role in communications systems. Man's capacity to handle auditory information (in the technical sense that this word has acquired since World War II) has been investigated in discrimination experiments that have ranged from "just noticeable differences" through "matching from a sample" to "absolute identification." In hearing, as in other sense modalities, we have witnessed the emergence[7] of a magical number as the most valid generalization for unidimensional stimulus continua.

(2) Emphasis on the informational aspects of speech perception has led to an extremely fruitful interaction with communication-oriented linguists,[8] who have contributed the key concept of "distinctive features" of speech sounds – a concept a parallel to which is still sadly lacking in the area of visual perception. Today's experimenters are no longer satisfied with gross articulation scores; instead, they are investigating speech as a multidimensional display; they are fitting their data into "confusion matrices" in order to describe the lawful ways in which speech communication deteriorates in the presence of distortion or noise.

(3) In the past, concern with the detectability of various sounds in the presence of noise had led to excellent and systematic data on masking. Lately, the influence of situational constraints (such as instructions, size and type of ensemble, "low-probability watch") upon the process of signal detection has been much studied; and we have been given data and models that attempt to quantify the way in which decision processes affect the values of such statistical invariants as detectable signal-to-noise ratios or even threshold.[9, 10]

(4) The post-World War II period has seen numerous studies of the two classical attributes of sound: loudness and pitch. The experiments on loudness reflect pressure of practical problems and progress in scaling methodology. It is probably too early to assess the far-reaching consequences for the field of audition of the recent work of Stevens.[11] Starting from a thorough investigation of the different ways of measuring loudness, he may well have scored a breakthrough in an area of classical psychophysics; in this area nonpsychologists are unfortunately familiar with one name only, that of Fechner – the mel scale for pure tones has not given rise to much controversy; pitch judgments for complex sounds (for example, repeated bursts of noise, or sounds that exhibit Schouten's "residue") continue to defy certain oversimplified theoretical views*.

*In a recent paper Békésy[12] takes issue with the common and misleading usage of the phrase, "theory (or theories) of hearing." These so-called theories, he says, are "usually concerned only with answering the question: 'How does the ear discriminate pitch?'" Thus, such theories of hearing should be more correctly identified as "basically theories concerning the vibration pattern of the basilar membrane and the sensory organs attached to it." A sophisticated account of the problem of auditory frequency analysis is given by Licklider.[13]

(5) With the advent of precise timing and phasing equipment, the problems of binaural interaction have attracted many experimenters. Time-honored topics such as monaural versus binaural threshold and auditory localization in space have been thoroughly reinvestigated; novel findings on interaural phase relations in masking have influenced our views on "critical bands" and have made it obvious that the two ears are wired together flexibly in the auditory nervous system.

(6) Finally, much curiosity has been exhibited in the temporal characteristics of the auditory system. Standard psychophysical data have been collected for stimuli that were carefully patterned along the time dimension. The exposure to intense noise in both industry and the military services has stimulated interest in recovery phenomena (including so-called "residual masking" and "poststimulatory fatigue"): audiometry à la Békésy and the time sequences of speech events have drawn attention to the dynamic operating characteristics of the auditory system; included here are studies on the validity of the $I \times t$ law, "perstimulatory fatigue," and "loudness adaptation."

Major Conceptual Trends that Have Influenced Research Workers in Audition

The advances in instrumentation, auditory physiology, and psychophysics that we have just outlined in skeleton fashion have taken place in a period in which the field has undergone a process of conceptual emancipation. Behind us lies the period in which most of the theoretical framework was furnished by classical physics, with its emphasis upon conservation laws and linear differential equations and with its basic model of the organism as a more or less passive transducer or filter.

The organism is now, in terms of prevalent jargons, a self-organizing computing machine, a coalition of maximum likelihood detectors, an actively decoding network that reaches selectively out into its environment. Past reinforcements shape the organism's response probabilities to the point where the stimulus ceases to be represented by a change in the here and the now. Sequential behavior and context have become proper objects of quantification. Probability theory and, in particular, information theory have left imprints upon the design of many recent experiments in audition.

During this last decade our conception of the nervous system has also changed dramatically. The cybernetically contaminated *Zeitgeist* is, in some disguises, successfully overcoming older views. According to these older views, the events that are basic to sensory communication take place in the end organ, and it was more or less implicitly assumed that they could be studied in anesthetized preparations. The use of novel techniques by Magoun and others has emphasized the role of the brain-stem reticular formation in behavior that relates to "attention" and

"habituation."[14,15] Although these workers have shown little interest in studying the influence of stimulus parameters upon the complex responses of various neural structures, they have done far more than just demonstrate a multiplicity of pathways. From these researches have emerged neuroanatomical hypotheses and, perhaps, even some of the physiological mechanisms[16,17] and principles (such as the principle of centrifugal control[18]) that may permit us to study meaningfully the ways in which an organism builds up discriminations or learns to handle the information that is neurally coded along its afferent pathway. Such behavior is not "evoked" in milliseconds. The electrophysiologist interested in sensory processes faces the need of developing techniques for the assessment of slow and persistent changes in spatiotemporal patterns.

At this point it would be appropriate to review in some detail several problems in audition by comparing relevant psychophysical and physiological data. Space is lacking to do more than to list some quasidogmatic summaries, together with some references.

(1) The "representation" of various kinds of pitch in the nervous system; the paradox according to which frequency discrimination is best, not at threshold (when presumably the least number of units is involved) or for high frequencies (when there is the sharpest localization in the mechanical vibration pattern); a statistical interpretation of this apparent paradox. The effect of cortical ablations upon frequency discrimination.[19]

(2) The "representation" (or representations) of intensity in the nervous system. A discussion of different intensity functions[20,21,22] that relate either frequency of firing or magnitude of response to stimulus intensity at various locations in the nervous system; perstimulatory fatigue and equilibration or adaptation.

(3) The effects of instructions (or judgment variables) upon psychophysical thresholds and upon the nervous system. Possible functions of the centrifugal pathways as (a) devices for the reduction of neural "noise" (effect upon spontaneous activity?), or (b) "tuning" controls of peripheral neural structures and perhaps even end organ-neural junctions, or (c) "gating" devices of incoming activity at various neural way stations, or more generally (d) as modifiers of statistical parameters (decision criteria) for populations of neurons.

(4) Just noticeable differences in relation to stimulus intensity; the role of neural "noise" near the absolute threshold; functional evidence for the presence of two neural populations with different properties; Y. Katsuki's work[23] on spontaneous activity in the lateral line organ of fish, and L. S. Frishkopf's data[24] on masking and amplitude variability in the so-called N_1-response to clicks.

(5) Problems of auditory localization and complex binaural interaction.[25,26] Discriminable interaural delays and variability of neural

latencies; electrophysiological evidence from anesthetized preparations; possibility that efferent pathways act as modifiers of mechanisms of binaural interaction.

(6) Behavioral decision or reaction times versus latencies of electrical responses in the nervous system: relation to informational aspects of sensory messages.[27]

Summary

Where do we stand with respect to physiologically oriented theories of hearing? A few years ago, Békésy[28] wrote, "For every level of anesthesia we will probably have a different hearing theory." Permit me to extend this limitation: we may also need a different "theory of hearing" for each level of *un*anesthesia, for various levels or kinds of learning or conditioning and perhaps even different theories to account for the behavior of the organism, depending on how much information is to be handled. We may thus feel that any attempt to predict, on the basis of our present physiological knowledge, the verbal or behavioral responses of even carefully selected, instructed, and properly motivated listeners is doomed, at best, to a rather limited success. Possibly we ought to conclude then that this type of theorizing – in the meaning that this phrase had in the first half of the twentieth century – is not particularly profitable.

There is, however, no reason to despair. Workers will continue to be interested in the physiological mechanisms that come into play when acoustic stimuli are presented to organisms. Workers will continue to formulate models and microtheories to account for specific sets of experimental data. However, the recognition is likely to grow that, in order to predict the way in which organisms will respond to auditory stimulation, we need more than a conventional "theory of hearing." We need theoretical views that are consonant with an over-all theory of behavior, albeit a physiologically oriented one. Such a theory will include an account of what happens at the sense organ and an account – in terms of our general understanding of brain function – of how the nervous system deals with sensory stimuli in the communicating animal.

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CONFERENCES HELD

SECTION OF BIOLOGY

PROTEIN NUTRITION*

Friday, March 1 and
Saturday, March 2, 1957

Conference Chairman: Fredrick J. Stare
Harvard University School of Public Health
Boston, Mass.

FRIDAY, MARCH 1, 1957

Session Chairman: Fredrick J. Stare
Harvard University School of Public Health
Boston, Mass.

9:00 A.M. –
Greetings from the Academy – A. S. Gordon, Chairman, Section of Biology, The New York Academy of Sciences, New York, N. Y.; New York University, New York, N. Y.

"Protein Needs of Children" – I. Genevieve Stearns, University of Iowa School of Medicine, Iowa City, Iowa.
II. Harold Stuart, Harvard University School of Public Health, Boston, Mass.

"Protein Needs of Adolescents" – Joseph A. Johnston, Henry Ford Hospital, Detroit, Mich.

Discussion:

Paul György, University of Pennsylvania School of Medicine, Philadelphia, Pa. (10 minutes)

Nathan Talbot, Massachusetts General Hospital, Boston, Mass. (10 minutes)

H. D. Lynch, Evansville, Ind. (10 minutes)

Jack Metcoff, Michael Reese Hospital, Chicago, Ill. (10 minutes)

J. K. Gallagher, Children's Hospital, Boston, Mass. (10 minutes)

*This conference was held in cooperation with E. I. Du Pont de Nemours & Company, Inc., Wilmington, Del.

"Protein Needs in Geriatrics" – Donald Watkin, United States Public Health Service, Bethesda, Md.

Discussion:

Margaret Ohlson, University of Iowa School of Medicine, Iowa City, Iowa. (10 minutes)

Frederic D. Zeman, The House for Aged and Infirm Hebrews, New York, N. Y. (10 Minutes)

Session Chairman: Nevin Scrimshaw
INCAP, Guatemala City, Guatemala, C. A.

2:00 P.M. –
"Prevention and Treatment of Kwashiorkor Disease" – Jean Sénécal, School of Medicine, University of Dakar, French West Africa.

"Prevention and Treatment of Kwashiorkor Disease" – Moisés Béhar, INCAP, Guatemala City, Guatemala, C. A.

"Prevention and Treatment of Kwashiorkor Disease" – Frederick Gomez, Children's Hospital Medical Center, Mexico City, Mexico.

Discussion:

Paul György, University of Pennsylvania School of Medicine, Philadelphia, Pa. (10 minutes)

H. E. Sauberlich, Alabama Polytechnic Institute, Auburn, Ala. (10 minutes)

Jack Metcoff, Michael Reese Hospital, Chicago, Ill. (10 minutes)

Nevin Scrimshaw, INCAP, Guatemala City, Guatemala. (10 minutes)

EVENING SESSION
(Open Session)
The New York Academy of Sciences
2 East 63rd Street

8:00 P.M. –
"Certain Biochemical Findings in Man as They Relate to Diet" – A. R. P. Walker, Director, Human Biochemical Unit, South African Institute for Medical Research, Johannesburg, South Africa.

SATURDAY, MARCH 2, 1957

Session Chairman: Fredrick J. Stare
Harvard University School of Public Health
Boston, Mass.

9:00 A.M. –

"Calories and Protein Utilization" – James B. Allison, Rutgers University, New Brunswick, N. J.

Discussion:

William Abbott, Western Reserve University, Cleveland, Ohio. (10 minutes)

T. B. Van Itallie, Harvard University School of Public Health, Boston, Mass. (10 minutes)

"Balance and Imbalance of Amino Acids" – Alfred Harper, University of Wisconsin, Madison, Wis.

Discussion:

James B. Allison, Rutgers University, New Brunswick, N. J. (10 minutes)

H. E. Sauberlich, Alabama Polytechnic Institute, Auburn, Ala. (10 minutes)

"Enrichment of Foods with Protein" – James Hundley, F. A. O., United Nations, New York, N. Y.

Discussion:

Nevin Scrimshaw, INCAP, Guatemala City, Guatemala, C. A. (10 minutes)

James B. Allison, Rutgers University, New Brunswick, N. J. (10 minutes)

General Summary

SECOND CONFERENCE ON SULFONAMIDES

Friday, March 22, 1957

Conference Chairman: Perrin H. Long
State University of New York School of Medicine
Brooklyn, N. Y.

Session Chairman: Maxwell Finland
Boston City Hospital, Boston, Mass.

9:00 A.M. –

Greetings from the Academy – A. S. Gordon, Chairman, Section of Biology,

The New York Academy of Sciences, New York, N.Y.; New York University, New York, N.Y.

Introduction of Gerhard Domagk, Farbenfabriken Bayer, Wuppertal-Elberfeld, Germany – Perrin H. Long, State University of New York School of Medicine, Brooklyn, N.Y.

Response by Gerhard Domagk

"Current Use of Sulfonamides in Internal Medicine" – Perrin H. Long, State University of New York School of Medicine, Brooklyn, N.Y.

"Current Use of Sulfonamides in Pediatric Practice" – Horace L. Hodes, Mount Sinai Hospital, New York, N.Y.

"The Clinical Use of Sulfisomidine" – A. M. Rutenburg, Beth Israel Hospital, Boston, Mass.

"Current Use of Sulfonamides in Urology" – Grayson L. Carroll, University of St. Louis, St. Louis, Mo.

"Clinical Use of a Lipid Emulsion of Sulfisoxazole" – Saul Krugman, New York University-Bellevue Medical Center, New York, N.Y.

"The Concurrent Use of Sulfonamides and Antibiotics in the Treatment of Infections" – Louis Weinstein, Boston University School of Medicine, Boston, Mass.

"Clinical Toxicity of Sulfonamides" – David Lehr, Flower & Fifth Avenue Hospitals, New York, N.Y.

Session Chairman: E. K. Marshall, Jr.
Johns Hopkins University School of Medicine
Baltimore, Md.

2:00 P.M. –

"Experimental Activities of New Sulfonamides" – Rudolf L. Mayer, CIBA Pharmaceutical Products, Inc., Summit, N.J.

"Experimental Investigations of Sulfamethoxypyridazine" – Raymond R. Roepke, Thomas H. Maren and Edmund Mayer, Lederle Laboratories Division, American Cyanamid Company, Pearl River, N.Y.

"Sulfamethoxypyridazine and Sulfachloropyridazine" – Wilfred F. Jones, Jr., and Maxwell Finland, Boston City Hospital, Boston, Mass.

"Clinical Use of Sulfamethoxypyridazine in Rheumatic Disease" – Mark H. Lepper, University of Illinois College of Medicine, Chicago, Ill.

"Clinical Use of Sulfamethoxypyridazine" – George G. Jackson, University of Illinois College of Medicine, Chicago, Ill.

"Clinical Use of Sulfamethoxypyridazine in Pediatrics" – Walter E. Ahrens, Children's Hospital, Washington, D.C.

"Clinical Use of Sulfamethoxypyridazine" – Morton Hamburger, University of Cincinnati College of Medicine, Cincinnati, Ohio.

"Clinical Use of Sulfachloropyridazine" – Robert C. Batterman, Bird S. Coler Memorial Hospital, New York, N.Y.

COMPARATIVE CLINICAL AND BIOLOGICAL EFFECTS OF ALKYLATING AGENTS

Thursday, March 28, Friday, March 29 and Saturday, March 30, 1957

Conference Chairman: Leon H. Schmidt
The Christ Hospital Institute of Medical Research
Cincinnati, Ohio

THURSDAY, MARCH 28, 1957

CHEMISTRY OF ALKYLATING AGENTS

Session Chairman: Norman H. Cromwell
University of Nebraska, Lincoln, Neb.

9:00 A.M. –
Greetings from the Academy – A. S. Gordon, Chairman, Section of Biology, The New York Academy of Sciences, New York, N.Y.; New York University, New York, N.Y.

Introductory Remarks – Leon H. Schmidt, The Christ Hospital Institute of Medical Research, Cincinnati, Ohio.

"Fundamental Mechanisms of Alkylation" – C. C. Price, University of Pennsylvania, Philadelphia, Pa.

"*In Vitro* Actions of Alkylating Agents" – W. C. J. Ross, Chester Beatty Research Institute, Royal Cancer Hospital, London, England.

"*In Vitro* Reactions of Alkylating Agents with Macromolecules" – K. A. Stacey, Chester Beatty Research Institute, Royal Cancer Hospital, London, England.

"Comparative *in Vitro* Effects of Alkylation and Radiation" – E. L. Powers, Argonne National Laboratory, Lemont, Ill.

Discussion: Norman H. Cromwell, University of Nebraska, Lincoln, Neb.

J. J. Roberts, Chester Beatty Research Institute, Royal Cancer Hospital, London, England.

G. M. Timmis, Chester Beatty Research Institute, Royal Cancer Hospital, London, England.

BIOLOGICAL EFFECTS OF ALKYLATING AGENTS (A)

Session Chairman: Alexander Haddow
Chester Beatty Research Institute, Royal Cancer Hospital
London, England

2:00 P.M. –
"Mutagenic Effects of Alkylating Agents" – C. Auerbach, Institute of Animal Genetics, Edinburgh, Scotland.

Discussion: O. G. Fahmy, Chester Beatty Research Institute, Royal Cancer Hospital, London, England.

Irwin Oster, University of Indiana, Bloomington, Ind.

"Carcinogenic Activity of Alkylating Agents" – Arthur L. Walpole, Imperial Chemical Industries, Ltd., Manchester, England.

Discussion: Alexander Haddow, Chester Beatty Research Institute, Royal Cancer Hospital, London, England.

"Effects of Alkylating Agents on Embryonic Development" – M. L. Murphy, Sloan-Kettering Institute for Cancer Research, New York, N. Y.

"Comparative Effects of Alkylating Agents on Cellular Morphology" – P. C. Koller, Chester Beatty Research Institute, Royal Cancer Hospital, London, England.

"Time and Localization of Chromosome Change and a Hypothesis of Action of Alkylating Agents" – S. H. Revell, Chester Beatty Research Institute, Royal Cancer Hospital, London, England.

Discussion: Howard E. Skipper, Southern Research Institute, Birmingham, Ala.

BIOLOGICAL EFFECTS OF ALKYLATING AGENTS (B)

Session Chairman: F. S. Philips
Sloan-Kettering Institute for Cancer Research
New York, N. Y.

8:00 P.M. –

"Pharmacological and Pathological Effects of Alkylating Agents" – S. S. Sternberg, Sloan-Kettering Institute for Cancer Research, New York, N. Y.

"Hematological Effects of the Alkylating Agents" – L. A. Elson, Chester Beatty Research Institute, Royal Cancer Hospital, London, England.

"Distribution and Fate of Alkylating Agents" – Paul K. Smith, George Washington University, Washington, D. C.

"Tumor-Inhibiting Effects of Alkylating Agents" – C. C. Stock, Sloan-Kettering Institute for Cancer Research, New York, N. Y.

Discussion: Alexander Haddow, Chester Beatty Research Institute, Royal Cancer Hospital, London, England.

Arthur L. Walpole, Imperial Chemical Industries, Ltd., Manchester, England.

Hugh J. Creech, Institute for Cancer Research, Philadelphia, Pa.

"General Biological Activities of Alkylating Agents (Summary)" – F. S. Philips, Sloan-Kettering Institute for Cancer Research, New York, N. Y.

FRIDAY, MARCH 29, 1957

CLINICAL RESULTS IN CANCER (A)

Session Chairman: Alfred Gellhorn
Columbia University College of Physicians and Surgeons
New York, N. Y.

9:00 A.M. –

"Necessary Criteria for the Comparative Evaluation of Alkylating Agents in Man" – Alfred Gellhorn, Columbia University College of Physicians and Surgeons, New York, N. Y.

"Summary of Results Obtained with Nitrogen Mustard" – David A. Karnofsky, Sloan-Kettering Institute for Cancer Research, New York, N. Y.

"Summary of Results Obtained with Chlorambucil" – I. G. Israels, University of Manitoba, Winnipeg, Man., Canada.

"Summary of Results Obtained with TEM" – Wayne Rundles, Duke University School of Medicine, Durham, N. C.

"Summary of Results Obtained with Thio-TEPA" – Jane Wright, New York University-Bellevue Medical Center, New York, N. Y.

"Summary of Results Obtained with Myleran" – David Galton, Chester Beatty Research Institute, Royal Cancer Hospital, London, England.

CLINICAL RESULTS IN CANCER (B)

Session Chairman: David A. Karnofsky
Sloan-Kettering Institute for Cancer Research
New York, N. Y.

2:00 P.M. –

Representatives of various institutions will be invited or may volunteer to discuss their clinical experiences with the various alkylating agents. The Organizing Committee will select the final program.

Louis K. Alpert, George Washington University Cancer Clinic, Washington, D. C.

Jeanne C. Bateman, Garfield Memorial Hospital, Washington, D. C.

Frank Bethell, University of Michigan Medical School, Ann Arbor, Mich.

W. H. Cole, University of Illinois, Chicago, Ill.

William Dameshek, Joseph H. Pratt Diagnostic Hospital, Boston, Mass.

H. D. Diamond, Memorial and James Ewing Hospitals and Sloan-Kettering Institute for Cancer Research, New York, N. Y.

Charles A. Doan, Ohio State University College of Medicine, Columbus, Ohio.

S. Farber, Children's Hospital, Boston, Mass.

Alfred Gellhorn, Columbia University College of Physicians and Surgeons, New York, N. Y.

S. Gumport, New York University-Bellevue Medical Center, New York, N. Y.

Stewart Jones, Lahey Clinic, Boston, Mass.

George Moore, Roswell Park Memorial Institute, Buffalo, N. Y.

K. B. Olson, Albany Medical College of Union University, Albany, N. Y.

A. Rottino, St. Vincent's Hospital, New York, N. Y.

Harry Shay, Fels Research Institute, Temple University School of Medicine, Philadelphia, Pa.

S. C. Shen, Tufts College Medical School, Boston, Mass.

R. D. Sullivan, Veterans Administration Hospital, New York, N. Y.

M. P. Sykes, Memorial and James Ewing Hospitals and Sloan-Kettering Institute for Cancer Research, New York, N. Y.

A. Weisberger, Western Reserve University, Cleveland, Ohio.

CLINICAL RESULTS IN CANCER (C)

Session Chairman: Gordon Zubrod
National Cancer Institute, National Institutes of Health
Bethesda, Md.

8:00 P.M. –

Experiences with more recent or unusual series of alkylating agents such as E-39, OMPSPA, chloroquine mustards, sarcolysin, hemi-sulfur mustards, alkylsulfonates (CB 1506 or chlorethylmethanesulfonate), dimethylmyleran, and others will be discussed by the following:

J. H. Burchenal, Memorial and James Ewing Hospitals and Sloan-Kettering Institute for Cancer Research, New York, N. Y.
David Galton, Chester Beatty Research Institute, Royal Cancer Hospital, London, England.
James Holland, Roswell Park Memorial Institute, Buffalo, N. Y.
Ralph Jones, University of Miami, Miami, Fla.
Yoshiyuki Koyama, Tokyo First National Hospital, Tokyo, Japan.
A. Seligman, Sinai Hospital, Baltimore, Md.
O. H. Warwick, Toronto General Hospital, Toronto, Ont., Canada.

SATURDAY, MARCH 30, 1957

SUMMARY DISCUSSION

Session Chairman: C. P. Rhoads
Sloan-Kettering Institute for Cancer Research
New York, N. Y.

9:00 A.M. –

"Relations Between Action of Radiation and the Alkylating Agents" – Peter Alexander, Chester Beatty Research Institute, Royal Cancer Hospital, London, England.

"Design of Alkylating Agents for Selectivity of Action" – F. Bergel, Chester Beatty Research Institute, Royal Cancer Hospital, London, England.

"Future Experimental Approaches" – Alexander Haddow, Chester Beatty Research Institute, Royal Cancer Hospital, London, England.

"Procedures Recommended for the Comparison of the Therapeutic Activity of Alkylating Agents in Man" – Gordon Zubrod, National Cancer Institute, National Institutes of Health, Bethesda, Md.

"Current Status of the Alkylating Agents in the Treatment of Cancer in Man" – Alfred Gellhorn, Columbia University College of Physicians and Surgeons, New York, N. Y.

SUMMARY OF CONFERENCE

C. P. Rhoads
Sloan-Kettering Institute for Cancer Research
New York, N. Y.

NEW MEMBERS

Elected March 28, 1957

SUSTAINING MEMBERSHIP

Bloom, Marvin L., M.D., Immunology. Associate Professor, Medicine, University of Buffalo School of Medicine, Buffalo, N. Y.

McKim, Mrs. Orville, General Sciences. Layman, New Rochelle, N. Y.

Reid, Duncan Earl, M.D., Human Reproduction. Professor, Harvard Medical School, Boston, Mass.

Van Winkle, Walton, Jr., M.D., Pharmacology. Director of Research, Ethicon, Inc., Somerville, N. J.

Weinstein, Howard, Ph.D., Physiology. Instructor, Biology Department, Wisconsin State College, Stevens Point, Wis.

Wilson, Robert B., M.D., Medicine. Member, Permanent Staff, Mayo Clinic, Rochester, N. Y.

Winks, Donald, Ph.B., Biology. Account Executive, Wm. Douglas McAdams, Inc., New York, N. Y.

Wright, Ernest Bevier, Ph.D., Neurophysiology. Associate Professor, University of Florida, Gainesville, Fla.

ACTIVE MEMBERSHIP

Agostino, Domenico, M.D., Biology. Assistant, Sloan-Kettering Institute for Cancer Research, New York, N. Y.

Altshuler, Charles H., M.D., Pathology. Pathologist, St. Joseph's Hospital, Milwaukee, Wis.

Andrews, John S., Ph.D., Foods. Director, Food Research, General Mills, Inc., Minneapolis, Minn.

Arteta, José Luis, M.D., Endocrinology. Associate Director, Experimental Institute of Endocrinology, Madrid, Spain.

Barnum, H. J., Jr., M.S., Food Technology. Manager, New Products, Lever Bros. Co., New York, N. Y.

Berger, Frederic William, M.D., Tropical Medicine. Research Consultant, Bronxville, N. Y.

Brunner, K. Theodor, D.V.M., Virology. Assistant Professor, New York University, New York, N. Y.

Cassin, Sidney, Ph.D., Physiology. Graduate Student, University of Texas, Galveston, Texas.

Cervoni, Peter, Ph.D., Autonomic Pharmacology. Lieutenant, U.S. Army, Jamaica, N. Y.

Chisena, Peter R., M.D., Medicine. Private Practice, Bridgeport, Mich.

Cohen, Irving J., B.S., Medical Sciences. Science Editor, New York, N. Y.

Collins, William E., Ph.D., Medical Entomology. Medical Entomologist, Fort Detrick, Frederick, Md.

Cornfeld, David, M.D., Biology. Director, Pediatric Clinic, Hospital of University of Pennsylvania, Bala-Cynwyd, Pa.

Cramblett, Henry Gaylord, M.D., Pediatrics. Clinical Research Associate, National Institutes of Health, Bethesda, Md.

Cranny, Robert LeVerne, M.D., Endocrinology. Instructor, Salt Lake County Hospital, Salt Lake City, Utah.

Daugherty, Jack W., Ph.D., Physiology. Associate Professor, Rice Institute, Houston, Texas.

Eckert, Ernst R.G., Dr. Ing., Thermodynamics. Professor, University of Minnesota, Minneapolis, Minn.

Edgecombe, S. W., Ph.D., Physics. Acting Provost, American University of Beirut, Beirut, Lebanon.

Elderfield, Robert C., Ph.D., Synthetic Chemistry. Professor, Chemistry, University of Michigan, Ann Arbor, Mich.

Elling, Dorothy Ann, B.A., Pharmacology. Assistant Pharmacologist, Nepera Chemical Company, Yonkers, N. Y.

Epstein, Norman, M.D., Allergy. Clinical Fellow, New York Medical College, New York, N. Y.

Eshaya, Allen M., Ph.D., Nuclear Engineering. Staff, Brookhaven National Laboratory, Upton, N. Y.

Feigen, George Alexander, Ph.D., Physiology. Associate Professor, Physiology, Stanford University, Stanford, Calif.

Florentin, Angel A., M.D., Endocrinology. Assistant Professor, Medicine, College of Medicine, University of the Philippines, Manila, Philippines.

Gerber, Paul, Ph.D., Virology. Head, Section of Virology, Squibb Institute for Medical Research, New Brunswick, N. J.

Glenn, E. Myles, Ph.D., Adrenal Cortical Steroid Physiology. Research Associate, The Upjohn Company, Kalamazoo, Mich.

Gochenour, William S., Jr., D.V.M., Microbiology. Chief, Animal Assessment Branch, U. S. Army Medical Unit, Fort Detrick, Frederick, Md.

Goe, Don R., Ph.D., Neurophysiology. Assistant Instructor, College of Medical Evangelists, Loma Linda, Calif.

Goldstein, Abraham, D.D.S., Dentistry. Assistant Professor, New York University College of Dentistry, Brooklyn, N. Y.

Golueke, Clarence G., Ph.D., Mycology. Research Biologist, University of California, San Pablo, Calif.

Goodner, John Tetard, M.D., Cancer. Clinical Assistant, Memorial Hospital, New York, N. Y.

Greathouse, R. F., M.D., Pediatrics. Pediatrician, Louisville, Ky.

Haffner, Alden Norman, O.D., Optometry. Director, Optometric Center of New York, Brooklyn, N. Y.

Harelick, Sally, B.A., Bacteriology. Biologist, Lederle Laboratories, Rockland Lake, N. Y.

Harris, Alvin E., M.D., Medicine. Private Practice, Shelley, Idaho.

Herrnstein, Richard J., Ph.D., Psychology. 1st Lt., M.S.C., U. S. Army, Silver Spring, Md.

Hicks, J. T., M.D., Biology. Staff, U. S. Public Health Service Hospital, Baltimore, Md.

Holt, C. Lawrence, M.D., Cardiology. Private Physician, Portland, Me.

Hursh, John B., Ph.D., Biophysics. Associate Professor, University of Rochester, Rochester, N. Y.

Krassner, Stuart, B.S., Parasitology. Graduate Student, Johns Hopkins University, Baltimore, Md.

Lein, Allen, Ph.D., Endocrinology. Associate Professor, Physiology, Northwestern University, Chicago, Ill.

Lennartz, H., M.D., Virology. Virologist, Institute of Multiple Sclerosis, Hamburg, Germany.

Lévy, Francois-Michel, M.D., Microbiology. Chief, Laboratories, International Children's Center, Paris, France.

Lombardo, Michael E., Ph.D., Biochemistry. Research Associate, College of Physicians & Surgeons, New York, N. Y.

Lynch, William G., M.D., Medicine. Associate, Attending Medicine, Wyckoff Heights Hospital, Glendale, N. Y.

McCain, B. E., M.D., Pathology. Staff, Veterans Administration Hospital, McKinney, Texas.

Moore, John W., Ph.D., Biology. Biophysicist, National Institutes of Health, Bethesda, Md.

Mulroy, Mary Irene, B.S., Endocrinology. Histologist, Nepera Chemical Company, Yonkers, N. Y.

Olson, J. Bennet, Ph.D., Embryology. Research Fellow, California Institute of Technology, Pasadena, Calif.

Osborne, Melville W., A.B., Mammalian Endocrinology. Graduate Student, Princeton University, Princeton, N. J.

Paine, John R., M.D., Surgery. Professor, Surgery, University of Buffalo, Buffalo, N. Y.

Palow, Alvin A., M.D., New Drugs. Internal Medicine, Industrial Physician, Armour Laboratories, Kankakee, Ill.

Peltier, Leonard F., Ph.D., Orthopedic Surgery. Professor, Surgery and Hea University of Kansas Medical Center, Kansas City, Kans.
Pette, Heinrich, Ph.D., Neurology. University of Hamburg, Hamburg, Germany.
Petranek, Joseph Robert, M.D., Biology. Clinical Fellow, Massachusetts General Hospital, Boston, Mass.
Pollock, Adrian Q., M.D., Pediatrics. Resident Physician, Pediatrics, Children's Hospital, Louisville, Ky.
Raw, Isaias, M.D., Biochemistry. Assistant Professor, Biochemistry, University of São Paulo, São Paulo, Brazil.
Regan, William M., A.M., Genetics. Professor, Animal Husbandry, University of California, Davis, Calif.
Reinhart, Justin H., Ph.D., Plant Physiology. Plant Physiologist, Olin Mathieson Corp., Port Jefferson Station, N. Y.
Reitemeier, R. F., Ph.D., Chemistry. Soil Scientist, U. S. Department of Agriculture, Beltsville, Md.
Richards, Ralph C., M.D., General Surgery. Assistant Professor, Surgery, University of Utah, Salt Lake City, Utah.
Rothenberg, Israel Roy, M.A., Microbiology. Microbiologist, North Shore Hospital, Levittown, N. Y.
Rudzki, Henryk S., Ph.D., Organic Chemistry. Director, Rexall Drug Company, Los Angeles, Calif.
Sage, Harold H., M.D., Tumors. Assistant Professor, Surgery, New York University College of Medicine, New York, N. Y.
Sears, Jack Wood, Ph.D., General Biology. Professor, Biology, Harding College, Searcy, Ark.
Shafiroff, Benjamin G. P., M.D., Cardiovascular. Associate Professor, New York University College of Medicine, New York, N. Y.
Shapiro, Max, D.D.S., Periodontia. Head, Periodontology Department, Cedars of Lebanon Hospital, Los Angeles, Calif.
Shaver, Arthur, Jr., M.D., Biology. Private Practice, Clinton, Md.
Sigg, Ernest B., M.D., Neurophysiology. Senior Physiologist, CIBA Pharmaceutical Products, Summit, N. J.
Simon, Raoul L., B.A., Biology. Biostatistician, Atomic Bomb Casualty Commission, San Francisco, Calif.
Simonsen, Daisy G., Ph.D., Cancer Research. Research Associate, City of Hope Medical Center, Duarte, Calif.
Skinner, Henry Alan Lawson, F.R.C.S., Anthropology. Professor, Anatomy, University of Western Ontario, London, Canada.
Slack, Keith V., Ph.D., Oceanography. Oceanographer, U. S. Navy Hydrographic Office, Washington, D. C.
Smith, Cecil G., M.S., Plant Pathology. Director, Agricultural Research, San Juan de Los Morros, Guarico, Venezuela.
Smith, Frank O., Ph.D., Plant Pathology. Associate Professor, American University of Beirut, Beirut, Lebanon.
Smith, Marshall E., M.D., Psychiatry. Clinical Director, Connecticut State Hospital, Middletown, Conn.
Solodar, Miriam R., M.A., Psychology. Speech Therapist, Newark Board of Education, Newark, N. J.
Sperling, Gladys A., M.S., Animal Nutrition. Research Associate, Cornell University, Ithaca, N. Y.
Stafford, Walter F., Jr., M.D., Neurology. Assistant Clinical Professor, Buffalo General Hospital, Buffalo, N. Y.
Starin, William A., Ph.D., Bacteriology. Professor Emeritus, Bacteriology, Ohio State University, Columbus, Ohio.
Starr, Leonard B., B.S., Experimental Pathology. Fellow, Department of Pathology, New York University College of Medicine, New York, N. Y.
Steele, J. Murray, M.D., Medicine. Professor, Medicine, Goldwater Memorial Hospital, New York, N. Y.
Stroman, G. N., Ph.D., Plant Breeding. Director, Institute of Genetics, National Agricultural Society, Lima, Peru.
Taliaferro, William H., Ph.D., Immunology. Professor, Microbiology, University of Chicago, Chicago, Ill

Thompson, Herman O., Ph.D., Pharmaceutical Forms. Professor, Pharmacy, University of North Carolina, Chapel Hill, N. C.

Thompson, I. Maclaren, M.B., Biometry. Professor, University of Manitoba, Winnipeg, Canada.

Thompson, R. E., Ph.D., Pharmacology. Head, Pharmacology Department, Armour Laboratories, Kankakee, Ill.

Thompson, Robert Q., Ph.D., Nutrition. Biochemist, Eli Lilly & Co., Indianapolis, Ind.

Thompson, T. C., M.D., Orthopedic Surgery. Chief Surgeon, Hospital for Special Surgery, New York, N. Y.

Thompson, Furness W., Ph.B., Administration of Research & Development. Vice President, Smith, Kline & French Laboratories, Philadelphia, Pa.

Thomson, J. R., M.S., Cancer Research. Biologist, Southern Research Institute, Birmingham, Ala.

Thornton, Wilmot A., Ph.D., Vertebrate Ecology. Associate Professor, Sam Houston Teachers College, Huntsville, Texas.

Threefoot, Sam A., M.D., Internal Medicine. Assistant Professor, Medicine, Tulane University, New Orleans, La.

Tidrick, R. T., M.D., General Surgery. Head, Department of Surgery, State University of Iowa College of Medicine, Iowa City, Iowa.

Tillman, Allen Douglas, Ph.D., Animal Nutrition. Research Participant, Oak Ridge Institute of Nuclear Studies, Oak Ridge, Tenn.

Tobias, Julian M., Ph.D., Neurophysiology. Professor, Physiology, University of Chicago, Chicago, Ill.

Tobie, John E., Ph.D., Immunology. Immunologist, National Institutes of Health, Bethesda, Md.

Tolman, Robert A., Ph.D., Zoology. Instructor, Des Moines Still College, Des Moines, Iowa.

Tomcufcik, Andrew S., Ph.D., Pharmaceutical Organic Chemistry. Group Leader, American Cyanamid Co., Pearl River, N. Y.

Tong, James Lun, Ph.D., Bacteriology. Bacteriologist, V.A. Hospital, Denver, Colo.

Tonhazy, Nicholas E., Ph.D., Cellular Physiology. Research Associate, Sterling-Winthrop Research Institute, Rensselaer, N. Y.

Toolan, Helene Wallace, Ph.D., Cancer. Associate, Sloan-Kettering Institute, New York, N. Y.

Towbin, Abe, M.D., Pathology. Associate Professor, State University of New York, New York, N. Y.

Travis, Dorothy F., Ph.D., Zoology. Assistant Professor, University of New New Hampshire, Durham, N. H.

Trotter, Mildred, Ph.D., Anatomy. Professor, Washington University, St. Louis, Mo.

Troyer, James R., Ph.D., Botany. Assistant Professor, Botany, North Carolina State College, Raleigh, N. C.

Tunison, Abram V., B.S., Fisheries. Chief, Branch of Game-Fish, U. S. Department of the Interior, Washington, D. C.

Turner, Clair E., Ph.D., Biology. Assistant to the President, National Foundation for Infantile Paralysis, Washington, D. C.

Turner, Naomi C., Ed.M., Biochemistry of Saliva. Research Associate, Forsyth Dental Infirmary for Children, Boston, Mass.

Tye, Russell, M.S., Chemist. Senior Research Associate, Kettering Laboratory, Batavia, Ohio.

Tylman, Stanley D., D.D.S., Dentistry. Professor and Department Head, College of Dentistry, University of Illinois, Chicago, Ill.

Uhle, Frederick C., Ph.D., Organic Chemistry. Assistant Professor, Harvard Medical School, Boston, Mass.

Ulfelder, Howard, M.D., Gynecology. Clinical Professor, Harvard Medical School, Boston, Mass.

Underkofler, Leland A., Ph.D., Biochemistry. Director, Research, Takamine Laboratory, Clifton, N. J.

Uyeyama, Kahn, M.D., Gastroenterology. Assistant Professor, Medicine, University of California, San Francisco, Calif.

Vaartaja, L. Olli, Ph.D., Biology. Pathologist, Canada Department of Agriculture, Saskatoon, Canada.

Vaccaro, Ralph Francis, M.P.H., Marine Biology. Research Associate, Woods Hole Oceanographic Institution, Woods Hole, Mass.

Valenstein, Elliot S., Ph.D., Psychology. Research Psychologist, Walter Reed Army Medical Center, Washington, D.C.

Van Buskirk, Charles, Ph.D., Clinical Neurology. Professor, University Hospital, Baltimore, Md.

VandeGrift, William Breen, Ph.B., Cytology. Director, Laboratories, St. Joseph's Hospital, Baltimore, Md.

Van Dyke, Donald Clair, M.D., Experimental Endocrinology. Research Physician, University of California, Berkeley, Calif.

Van Epps, Eugene F., M.D., Cardiovascular Radiology. Professor, Iowa State University Hospital, Iowa City, Iowa.

Van Maanen, Evert F., Ph.D., Pharmacology. Associate Director, Wm. S. Merrell Co., Cincinnati, Ohio.

Van Pelt, Arnold F., Ph.D., Ecology. Professor, Biology, Tusculum College, Greeneville, Tenn.

Van Volkenburgh, Vivian A., Ph.D., Public Health. Assistant Commissioner, New York State Department of Health, New York, N. Y.

Vaughn, Moses W., Ph.D., Food Technology. Professor, Maryland State College, Princess Anne, Md.

Vergeer, Teunis, Ph.D., Biology. Assistant Professor, University of North Dakota, Grand Forks, N. Dak.

Verma, Indrapal S., Ph.D., Biochemistry, Associate Professor, Lincoln University, Jefferson City, Mo.

Visscher, Maurice B., Ph.D., Biology. Professor, University of Minnesota, Minneapolis, Minn.

Wade, Preston A., M.D., Surgery. Professor, Cornell University Medical Center, New York, N. Y.

Wagner, Robert P., Ph.D., Genetics. Professor, Zoology, University of Texas, Austin, Texas.

Wald, Samuel Stanley, F.A.C.D., Radiology. Clinical Professor, New York University College of Medicine, New York, N. Y.

Walker, Ernest P., Mammalogy. Retired, Washington, D.C.

Walker, Harvey, Jr., M.D., Diabetes. Resident, Internal Medicine, Jewish Hospital of St. Louis, St. Louis, Mo.

Wallbrunn, Henry M., Ph.D., Genetics. Assistant Professor, Biology, University of Florida, Gainesville, Fla.

Ward, Robert, M.D., Biology. Professor, Pediatrics, New York University College of Medicine, New York, N. Y.

Ware, Arnold G., Ph.D., Biochemistry. Head Biochemist, Los Angeles County Hospital, Los Angeles, Calif.

Warner, Edward N., Ph.D., Biology. Acting Chairman, St. Lawrence University, Canton, N. Y.

Watanabe, Shizuo, D.Sc., Biochemistry. Research Associate, Georgetown University, Washington, D. C.

Watts, Malcolm S. M., M.D., Internal Medicine. Assistant Clinical Professor, University of California, San Francisco, Calif.

Weber, George R., Ph.D., Microbiology. Research Microbiologist, National Distillers Products Corp., Cincinnati, Ohio.

Weber, Patricia R., M.S., Genetics. Junior Literature Scientist, Smith, Kline & French Laboratories, Philadelphia, Pa.

Weikel, John H., Jr., Ph.D., Pharmacology. Senior Pharmacologist, Mead, Johnson & Co., Evansville, Ind.

Weil, Paul G., Ph.D., Hematology. Assistant Physician, Royal Victoria Hospital, Montreal, Canada.

Weinmann, Joseph P., M.D., Pathology. Professor, College of Dentistry, University of Illinois, Chicago, Ill.

Weinstein, Irwin M., M.D., Hematology. Chief, Medicine, Wadsworth General Hospital, Los Angeles, Calif.

Weintraub, Marvin, Ph.D., Plant Pathology. Senior Plant Pathologist, University of California, Berkeley, Calif.

Weir, John M., Ph.D., Medical Education. Associate Director, Rockefeller Foundation, New York, N. Y.

Wendel, Herbert A., M.D., Pharmacology. Senior Pharmacologist, Smith, Kline & French Laboratories, Philadelphia, Pa.

Werner, Henry James, Ph.D., Histocytology. Associate Professor, Louisiana State University, Baton Rouge, La.

Wesson, Laurence G., Ph.D., Biochemistry. Staff Member, Massachusetts Institute of Technology, Cambridge, Mass.

Wharton, P. S., M.A., Biochemistry. Professor, Austin College, Sherman, Texas.

Wheeler, Elizabeth S., Ph.D., Cytology. Supervisor, Cytology Laboratory, Hartford Hospital, Hartford, Conn.

Whitaker, Donald Robert, Ph.D., Biochemistry. Associate Officer, National Council of Research, Ottawa, Canada.

Whitaker, Ellis Hobart, Ph.D., Plant Physiology. Associate Professor, State University Teachers College, Oneonta, N. Y.

White, Edith Grace, Ph.D., Elasmobranch Fishes. Professor, Biology, Wilson College, Chambersburg, Pa.

White, Elizabeth Lloyd, Ph.D., Embryology. Associate Professor, Biology, Wheaton College, Norton, Mass.

White, Richard P., Ph.D., Neuropharmacology. Instructor, University of Tennessee Medical Units, Memphis, Tenn.

Whitelaw, Donald Mackay, F.R.C.P., Hematology. Associate Professor, University of British Columbia, Vancouver, B.C.

Whittaker, Robert H., Ph.D., Synecology. Instructor, Brooklyn College, Brooklyn, N. Y.

Williams, Henry, Jr., M.D., Clinical Cardiopulmonary Physiology. Director, Grasslands Hospital, Valhalla, N. Y.

Wilson, Norman J., M.D., Surgery. Surgeon, Overholt Thoracic Clinic, Boston, Mass.

Wirts, Charles Wilmer, M.D., Gastroenterology. Associate Professor, Jefferson Medical College, Philadelphia, Pa.

Wiseman, Bruce Kenneth, M.D., Hematology. Professor, Ohio State University, Columbus, Ohio.

Wistrŏm, Svante, Pharmacy. Director, Medical & Research Departments, AB Leo, Hälsingborg, Sweden.

Witten, Benjamin, Ph.D., Chemical Warfare. Chief, Chemical Warfare Laboratories, Army Chemical Center, Md.

Wittler, Ruth Graeser, Ph.D., Bacteriology. Bacteriologist, Walter Reed Army Institute of Research, Silver Spring, Md.

Womack, C. Ray, M.D., Infectious Diseases. Assistant Professor, Louisiana State University, New Orleans, La.

Womack, Nathan A., M.D., Surgery. Head, Department Surgery, University of North Carolina, Chapel Hill, N. C.

Worth, C. Brooke, M.D., Epidemiology. Research Associate, University of Pennsylvania, Philadelphia, Pa.

Wroblewski, Felix, M.D., Biochemistry. Assistant, Sloan-Kettering Institute, New York, N. Y.

Wu, Szu-Hsiao, Ph.D., Physiology. Instructor, Oregon State College, Corvallis, Ore.

Wynne, Edwin A., M.S., Analytical Chemistry. Director, Fisher Scientific Company, Fair Lawn, N. J.

Yarwood, C. E., Ph.D., Biology. Professor, Plant Pathology, University of California, Berkeley, Calif.

Yntema, Chester L., Ph.D., Biology. Professor, Anatomy, State University of New York, Syracuse, N. Y.

Yoder, Donald M., Ph.D., Plant Pathology. Head, Carbide & Carbon Chemicals Company, Yonkers, N. Y.

Young, Harold, M.S., Biochemistry. Chief Chemist, Consolidated Products Company, Danville, Ill.

STUDENT MEMBERSHIP

Freedman, Arlene, Physical Therapy. Student & Medical Assistant, Hunter College, New York, N. Y.

Mathews, Micheline Mary, B.S., Microbiology. First Year Medical Student, New York University, New York, N. Y.
Milstein, Sandra, Psychology. Student, Hunter College, New York, N. Y.
Rubin, Arnold David, B.A., Biochemistry. Student, New York University, New York, N. Y.
Shorr, Joshua, Psychology. Senior, Hunter College, New York, N. Y.

Elected April 25, 1957

SUSTAINING MEMBERSHIP

Calhoun, John Alfred, M.D., Medicine. Medical Director, American Viscose Corporation, Philadelphia, Pa.
Downs, Wilbur G., M.D., Medical Virology. Staff, The Rockefeller Foundation, Port of Spain, Trinidad.
Eckstein, Vincent Francis, Ph.D., Chemistry. Director, Technical Services, Merck Sharp & Dohme, Rahway, N. J.
Wagoner, George, M.D., Pathology. Professor, Orthopedic Surgery, University of Pennsylvania, Philadelphia, Pa.
Worzniak, Joseph J., M.D., Cancer. Private Practice, Wyandotte, Mich.

ACTIVE MEMBERSHIP

Andelman, Julian Barry, A.B., Physical Chemistry. Research Fellow, Polytechnic Institute of Brooklyn, Brooklyn, N. Y.
Araujo, Jorge, M.D., Pathology. Professor, Medicine, Universidad Del Valle, Colombia, S. A.
Barton, W. Bruce, M.D., Hematology. Physician, Ontario Cancer Foundation, London, Ont., Canada.
Bernstein, Jay, M.D., Pathology. Assistant Pathologist, Children's Hospital of Michigan, Detroit, Mich.
Blockus, Leo E., Jr., M.S., Pharmacology. Research Pharmacologist, Abbott Laboratories, North Chicago, Ill.
Bloom, Barry M., Ph.D., Medicinal Chemistry. Research Chemist, Chas. Pfizer & Co., Inc., Brooklyn, N. Y.
Bo, Walter J., Ph.D., Endocrinology. Assistant Professor, Anatomy, University of North Dakota, Grand Forks, N. Dak.
Bouroncle, Bertha A., M.D., Hematology. Assistant Professor, University Hospital, Columbus, Ohio.
Calkins, Harmond Eldred, Ph.D., Immunology. Associate Professor, Bacteriology, South Dakota State College, Brookings, S. Dak.
Calvo-Nuñez, Haroldo, M.D., Pathology. Professor, Universidad de Cartagena, Colombia, S. A.
Campbell, Gilbert Sadler, Ph.D., Surgery. Assistant Professor, University of Minnesota Hospital, Minneapolis, Minn.
Campbell, L. Leon Jr., Ph.D., Bacteriology. Assistant Professor, Washington State College, Pullman, Wash.
Campbell, Thomas H., Ph.D., Mycology. Assistant Professor, Botany, University of Tennessee, Knoxville, Tenn.
Cardon, Bartley P., Ph.D., Chemistry. Nutritionist, Arizona Flour Mills, Tucson, Ariz.
Carlson, Helen B., M.D., Psychiatry. Associate, Northwestern University Medical School, Winnetka, Ill.
Carlton, Harry N., M.D., Cancer. Private Practice, Washington, D.C.
Caro, Marcus Rayner, M.D., Dermatology. Professor, University of Illinois, Winnetka, Ill.
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